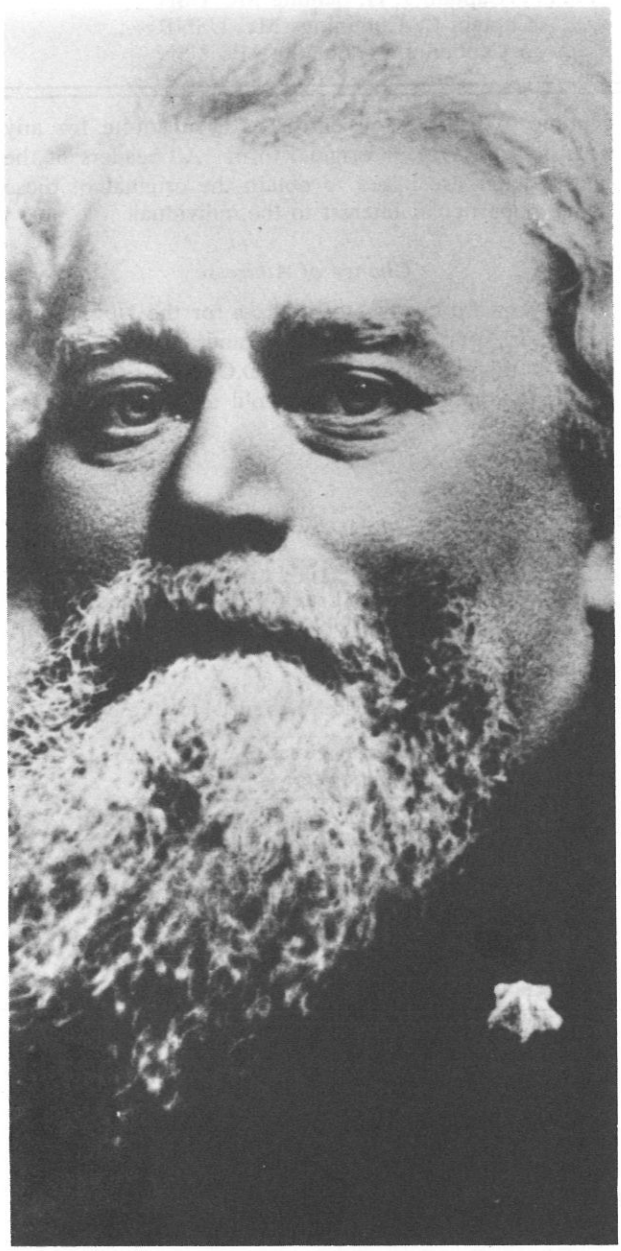
NAVMED P-5088

UNITED STATES NAVY
# Medical News Letter

Vol. 49 Friday, 10 February 1967 No. 3



Surgeons General of the Past
(The fifth in a series of brief biographies)

Doctor William Maxwell Wood was born 27 May 1809 in Baltimore, Md., was graduated from the University of Maryland School of Medicine, and was commissioned an Assistant Surgeon in the U.S. Navy 16 May 1829. His unusual, colorful, and varied service career included duty aboard vessels involved in the suppression of piracy and interruption of the slave trade, as well as activities in the Seminole Indian War, the Mexican War and the Civil War. As Fleet Surgeon of the Pacific Fleet he furnished American naval authorities with the earliest possible information on the outbreak of the Mexican War, thus enabling the Navy to seize California for the United States. He was aboard the USS SAN JACINTO when the East Indian Squadron arranged commercial relations with Siam and negotiated for trading ports with China and Japan in 1853. He was attached to a three-vessel squadron which protected American interests against Chinese anti-foreign feeling at Canton in 1856, being present during the attack on and destruction of the Cantonese forts. From 1862 to 1864 he served as Fleet Surgeon for a blockading squadron, attached to the flagship USS MINNESOTA. On 1 July 1869, he was appointed Chief, Bureau of Medicine and Surgery by President Grant. He became the first occupant of the office to be designated Surgeon General when that title was prescribed in an appropriation act of 3 March 1871 which also gave him the rank of Commodore. Doctor Wood was placed on the Retired List 27 May 1871 but served as Surgeon General until 25 October of that year. During his administration legislation was passed which grouped medical officers in a separate and distinct staff corps with grades established by law; and the naval hospital at Mare Island, California was built. On 1 April 1873 he went into full retirement, and he died at his home in Owing Mills, Baltimore County, Md., on 1 March 1880.

*United States Navy*
*MEDICAL NEWS LETTER*

Vol. 49 Friday, 10 February 1967 No. 3



*Policy*

The U.S. Navy Medical News Letter is basically an official Medical Department publication inviting the attention of officers of the Medical Department of the Regular Navy and Naval Reserve to timely up-to-date items of official and professional interest relative to medicine, dentistry, and allied sciences. The amount of information used is only that necessary to inform adequately officers of the Medical Department of the existence and source of such information. The items used are neither intended to be, nor are they, susceptible to use by any officer as a substitute for any item or article, in its original form. All readers of the News Letter are urged to obtain the original of those items of particular interest to the individual.

*Change of Address*

Please forward changes of address for the News Letter to Editor: Bureau of Medicine and Surgery, Department of the Navy, Washington, D.C. 20390 (Code 18), giving full name, rank, corps, old and new addresses, and zip code.

## CONTENTS



The issuance of this publication approved by the Secretary of the Navy on 4 May 1964.

# SPECIAL ARTICLE

## THE ARMED FORCES INSTITUTE OF PATHOLOGY: AN EDUCATIONAL OPPORTUNITY

*CAPT Bruce H. Smith MC USN, Deputy Director, Armed Forces Institute of Pathology, Washington, D.C. 20305.*

Founded in 1862 by Administrative Order of Surgeon General William Hammond of the U.S. Army, the Armed Forces Institute of Pathology was known until 1946 as the Army Medical Museum. From 1946 until 1949 it bore the name Army Institute of Pathology, and from 1949 to the present, it has operated under a Department of Defense charter as the Armed Forces Institute of Pathology.

Through its 105-year history the Armed Forces Institute of Pathology (AFIP) has had six homes, and has grown steadily into the current complex of four buildings, the primary one being located on the grounds of Walter Reed Army Medical Center. In accordance with its charter, it is a triservice organization operated under the Department of Defense with the Surgeons General of the Army, Navy, and Air Force constituting the Board of Governors. Its present director, MGEN Joe M. Blumberg MC USA, supported by Navy and Air Force deputies, commands what is probably the most unique staff ever assembled in one medical organization.

The triservice effort is quite ably supported not only by Army, Navy, and Air Force but also by Veterans Administration and Public Health Service personnel, as well as civilians from both governmental and nongovernmental sources and foreign countries.

It is this unique blend of pathologists and clinicians working side by side with basic scientists and auxiliary personnel that has firmly established the international reputation of the AFIP in the fields of consultation, education, and research. The acquisition of specimens from various sources through its consultation service has built the largest single repository of pathologic material ever assembled by one institution. In this massive collection of almost one and a half million accessions can be found examples of almost every pathologic condition known in man and many lower animals. It is upon this repository that the educational and research activities are based. The Institute's research efforts in many fields in this area of anatomic pathology are well known. Its efforts in this regard embrace many unique capabilities and employ up-to-date techniques such as electron microscopy, fluorescence microscopy, lasers, x-ray diffraction, and immunochemistry.

The educational facilities are voluminous and constitute one of the largest single postgraduate medical education programs in the world. Much of this has been due to the support of civilian medical, dental, and veterinary societies through the American Registry of Pathology, one of the four major departments of the AFIP. The Registry is responsible for the centralization of material from within and outside the Federal Government into a unique repository to be used by both pathologists and clinicians. Although its name indicates that the AFIP is a pathology institute, it is frequently forgotten hat this discipline of medicine is common to all specialties, and therefore approximately 70 percent of the educational product at the AFIP is utilized by clinicians seeking knowledge of pathology in their various clinical specialties.

The educational facilities at the AFIP can be divided into two categories: (A) in-house training, in which the student is required to come to the Institute for varying periods of time; and (B) training by mail, in which the educational material is forwarded to the student who is unable to make the trip to Washington.

*A. In-house training.*

1. Fellowships. The Institute offers fellowships of varying duration in the pathology of practically all clinical specialties. Many of these are supported by

an individual medical society providing stipends for nonmilitary personnel.

2. Residencies. Residencies are offered in neuropathology, forensic pathology, and general pathology on a fourth-year level. Although these are primarily for military personnel, residencies in neuropathology and forensic pathology are offered to civilian personnel as well, on a second-priority basis.

3. Formal courses. Each year the AFIP conducts between 17 and 22 formal courses on subjects designed primarily for the practicing clinical specialist. These run from 3 days to as long as 6 weeks. The courses are changed each year in accordance with the demand. Many are regarded as the best or the only course available on a particular specialty. In addition, through the American Registry of Pathology, a 3-month course is offered in radiologic pathology, correlating gross pathology with roentgenograms.

4. Symposia. Each year the Institute conducts two or three closed-invitational symposia on advanced subjects. Examples of recent years are symposia on the pathology of the audiovestibular apparatus, advances in leprosy, biologic effects of the laser, and quantitative electron microscopy.

5. Individual study. In addition to the more formal in-house programs, the Institute makes available an individual study program that permits physicians to come to the Institute for short periods of time, primarily to review material on an individual basis, usually in preparation for examination by various American specialty boards. This can be accomplished either in the individual branch or in specialized study rooms provided for visiting students.

6. Medical Museum. The AFIP also operates a medical museum, located on the Mall at 7th Street and Independence Avenue, S.W. This building houses a museum for the general public and a pathology museum available to professional visitors.

*B. Training by mail.*

Through the American Registry of Pathology and the Medical Illustration Service, the AFIP makes available numerous training aids that account for more than 26,000 loans each year. This educational material consists of motion pictures, lantern slide sets, microscopic slide sets, combination lantern and microscopic slide sets, and clinicopathologic conferences. These are available free of charge on a 2-week loan basis.

1. Motion-picture films. The Institute currently has 2,393 motion-picture films in its library. Although most of these films are on subjects dealing with pathology, many pertain to other related medical fields.

2. Lantern-slide sets. The present library contains 1,443 2 x 2-inch lantern-slide sets on 155 different subjects. Many of these are basic review sets designed especially to help the clinical specialist in his preparation for specialty-board examination. Each set is accompanied by a syllabus giving the necessary background, titles, and discussion.

3. Microscopic-slide sets. This portion of the library contains 2,798 slide sets under 161 titles. Although most of the sets contain 100 glass microscopic slides, several are combination sets containing not only microscopic slides but lantern slides showing the gross and x-ray aspects of the cases. Each set is accompanied by a syllabus containing a discussion of the cases.

4. Clinicopathologic conferences. The lending library has 2,109 CPC's under 642 titles, each of which is a complete case report. Each CPC contains a clinical summary, pathologic findings, discussion, and references. Also in the folder are 2 x 2 Kodachrome photographic transparencies illustrating clinical, gross, microscopic, x-ray, and other features of importance.

5. Video tapes and kinescopic recordings. Through the Walter Reed Army Medical Center Television Service, the AFIP has amassed a large number of video tapes on lectures given at the Institute. The AFIP is now in process of converting many of these into kinescopic recordings to be added to the lending library of motion pictures. In addition, selected prints of video tapes can be procured through special arrangement with the National Naval Medical Center.

6. Printed material. The AFIP operates two presses and is responsible for the production of many publications, including the Fascicles of the Atlas of Tumor Pathology, atlases, syllabi, monographs, technical manuals, and radiologic pathology workbooks. These are sold at cost through the American Registry of Pathology. In addition, reprints of articles written by staff members and published in medical journals can be procured free on a first-come, first-served basis as long as the supply lasts.

The educational facilities of the Institute are rapidly expanding, and up-to-date information pertaining to these training aids may be secured by writing to The Director, Armed Forces Institute of Pathology, Washington, D.C. 20305.

# MEDICAL ARTICLES

## SERUM ENZYME DETERMINATIONS IN DIAGNOSIS OF ISCHEMIC HEART DISEASE

### PITFALLS IN APPLICATION

*E. Boszormenyi MD,* F. Utsu MD,* V. Enescu MD,* H. Bernstein MD,† and E. Corday MD,‡ (University of California School of Medicine and Cedars-Sinai Medical Center, Los Angeles.)* *Postgrad Med 40(4):418–424, October 1966.*

*The SGOT test is said to have greater sensitivity than electrocardiography in the diagnosis of myocardial infarction. The LDH test is of particular aid when testing is started several days following the infarction. The temporal relationship between infarction and enzyme elevations has an important bearing on the determinations. Consideration must also be given to the many extracardiac factors that affect the enzyme levels.*

Serum enzyme determinations are now established as valuable aids in the diagnosis of infarction of the myocardium. Indeed, enzyme elevations have proved to be more effective in this diagnosis than electrocardiographic infarction patterns. The basis for diagnostic use of serum enzyme determinations is liberation of the enzymes from injured or ischemic myocardial tissue into the plasma. However, extracardiac factors may also affect serum enzyme levels. Conditions other than myocardial infarction which cause elevations of serum enzymes will be enumerated in this report.

LaDue, Wroblewski and Karmen first established the diagnostic value of the serum glutamic-oxalacetic transaminase (SGOT) and lactic acid dehydrogenase (LDH) tests in determining myocardial tissue injury. Tests for these two enzymes, which are the principal ones whose levels are increased in the serum following myocardial injury, have been the most useful for diagnostic purposes. However, many other enzymes may also be freed by necrosis of the myocardium. The serum creatine phosphokinase (CPK) and alpha-hydroxybutyrate dehydrogenase (HBD) also become elevated following myocardial infarction. Other enzymes are present in the serum in substantial amounts but their practical diagnostic significance has not been established.

Serum enzymes are not released exclusively by myocardial tissue. Therefore, these determinations cannot be used as specific tests for myocardial infarction. Proper interpretation of the results requires a knowledge of the various extracardiac factors which can cause an elevation of the enzymes.

#### Serum Glutamic-Oxalacetic Transaminase

It is believed that the SGOT becomes elevated in 97 percent of cases of myocardial infarction. In a study of 2,227 patients with clinical evidence of myocardial infarction the SGOT was found to be elevated in 97 percent. In a series of 1,255 cases of proved myocardial infarction the test gave false negative results in 3 percent. Elevation of SGOT was recorded in 137 of 142 cases of myocardial infarction proved at autopsy. LaDue, Wroblewski and Karmen demonstrated that the SGOT level begins to rise within 24 hours following coronary occlusion and remains elevated for a period ranging up to three days. Wroblewski and LaDue as well as Chinsky and Sherry have indicated that the normal value for SGOT ranges from 5 to 40 units per milliliter. Whetzel described a rapid screening test.

Meyers and Evans compared the diagnostic capabilities of the SGOT test and electrocardiography in 157 cases of myocardial infarction proved at autopsy. The SGOT was elevated in 97 percent whereas the electrocardiograms were diagnostic in 81.5 percent. These observations suggest that the SGOT test has a greater sensitivity than the electrocardiogram in the diagnosis of myocardial infraction.

* Research Fellow, Medical Research Institute, Cedars-Sinai Medical Center; †Clinical Instructor in Medicine, University of California School of Medicine; ‡Associate Clinical Professor of Medicine, University of California School of Medicine, Los Angeles.

At one time it was believed that the size of a myocardial infarction could be judged by the degree of elevation of the SGOT. However, associated ischemia of organs served by the systemic circulation can cause liberation of the enzymes from remote organs such as the liver. When shock supervenes and causes liver ischemia, therefore, the level of these enzymes does not truly reflect the size of the myocardial infarction.

Lactic Dehydrogenase

It is believed that the LDH becomes elevated in 95 percent of cases of myocardial infarction. It has been demonstrated that LDH actually constitutes a series of enzymes. Lactic acid dehydrogenase, an enzyme of the glycolytic cycle, reversibly catalyzes the conversion of pyruvate to lactate, utilizing diphosphopyridine nucleotide (DPNH, DPN) as an oxidizable-reducible coenzyme for the reaction.

The serum LDH level rises during the second or third day following myocardial infarction, reaches a peak on the fourth or fifth day, and then descends. If this test is used to detect myocardial infarction, therefore, the blood will have to be collected at a time when the enzyme level is most likely to be elevated. Bang and LaDue stated that the LDH determination is not more reliable than the SGOT test in the diagnosis of myocardial infarction except when the patient is first seen later in the course of the infarct. The LDH test can be used to confirm the SGOT elevation. Also, because the LDH level reaches a peak later than the SGOT, it is of particular benefit if testing is started several days following the myocardial infarction, when the SGOT level has already descended.

Wieland and Pfleiderer demonstrated that LDH from tissues could be separated into isozymes with species-specific and organ-specific electrophoretic patterns. Plagemann, Gregory and Wroblewski identified five LDH isozymes in rabbit and human tissues and observed typical patterns for each tissue. Other investigators have corroborated these observations. The LDH in heart muscle is relatively rich in the fast-moving isozymes. Therefore the electrophoretic pattern of the serum LDH exhibits a preponderance of these fractions after cardiac infarction. Elliott, Jepson and Wilkinson demonstrated that the fast-moving isozymes have relatively high hydrogenase activity with 2-oxobutyrate as the substrate, whereas the slow fractions showed comparatively little activity with this substrate. Elliott and Wilkinson reported that determination of the 2-hydroxybutyrate dehydrogenase (HBD) using 2-oxobutyrate as substrate is of considerable diagnostic value in myocardial infarction.

It is stated that the serum HBD-LDH ratio has the utmost importance in detecting myocardial infarction. The ratio usually rises above the normal upper limit of 0.81. Liver LDH consists largely of the slowest isozyme, LDH. The HBD-LDH ratio of liver extract is thus about 0.33. The serum HBD is said to be more specific than other enzyme tests.

Heat fractionation has been utilized in attempts to refine the LDH determinations. It seemed that the LDH from human heart muscle should be more heat-stable than LDH from other tissues. Bell stated the belief that activity of serum LDH in excess of 85 units after incubation at 60° C for 60 minutes is diagnostic of myocardial infarction.

Determination of total LDH activity is considered to be chiefly a screening test. The discovery of LDH isozymes presents new possibilities for testing specific organ ischemia.

Glutamic-Pyruvic Transaminase (GPT)

The content of this enzyme in the myocardium is rather small, and the GPT test is therefore of little value in the diagnosis of myocardial infarction. The serum level of the enzyme will not rise significantly unless secondary ischemia due to cardiogenic shock or arrhythmia causes it to be liberated from remote organs. The pyruvic transaminase test is more sensitive in depicting acute hepatocellular damage and is less sensitive as an indicator of acute myocardial necrosis. The myocardium is approximately 20 times richer in oxalacetic transaminase than in pyruvic transaminase. The normal serum pyruvic transaminase level ranges from 5 to 30 units per milliliter.

Creatine Phosphokinase

Study of the serum CPK activity was begun in 1959 by Ebashi and associates. Increased levels of CPK in the serum of patients with acute myocardial infarction were first reported by Dreyfus and coworkers. CPK elevation was believed to reflect ischemia of the myocardium more clearly than elevation of other enzymes because of a greater specificity of the test for acute myocardial necrosis and because the enzyme has a low activity in other areas (erythrocytes, lung, liver, kidney, pancreas).

Hess and associates and Vincent and Rapaport found that the serum CPK level regularly rose early in the course of acute myocardial infarction but also encountered elevations in the presence of damage to

brain and skeletal muscle. In contrast, patients with disease of the liver and lung consistently had normal serum CPK values. Normal levels were observed in congestive heart failure, rapid supraventricular tachycardia or arrhythmias, and acute coronary insufficiency without evidence of infarction. There is a transient increase in the serum activity of CPK in normal subjects after moderately severe exercise. The upper limit of normal for serum CPK, determined by the method of Tanzer and Gilvarg, is 2 to 3 units per milliliter. Serial determinations reveal that the serum enzyme level becomes elevated as early as six hours following its release and remains elevated through the third day. Elevation following myocardial infarction has been confirmed.

### Timing of the Tests

Because of the temporal relationship between myocardial infarction and elevation of the enzymes, blood samples for the tests must be taken at the most advantageous time. In selecting the test to be performed, the physician should consider the probable time of occurrence of the infarction. If the investigation is started several days after the infarction, the serum GOT and CPK will have returned to normal levels and only the LDH test will provide diagnostically helpful information.

### Significance of Fixed Elevation

SGOT and LDH elevations revert to a base line within about a week following myocardial infarction in the absence of other conditions causing an increase in the enzyme levels. There are many disease states, such as hepatitis and cancer, in which the levels do not descend. Fixed enzyme elevations therefore usually indicate that the increases are due to other causes.

### Sources of False Test Results

*Drugs*—Many drugs have been found to cause false positive serum enzyme tests. Enzyme tests are very sensitive to drug-induced liver dysfunction and are therefore an excellent means of demonstrating drug-induced parenchymal hepatitis.

*Laboratory error*—Because different laboratories may employ different technics and standards in the performance of the serum enzyme tests, all testing in a given case should be done in the same laboratory. Failure to use a constant-temperature apparatus to warm the stage on which the test tube is examined may lead to error; there is a 7 percent variation in the SGOT reading per degree of temperature. False positive results may be obtained if the blood is allowed to stand too long or if the test tube is wet. Only 10 mg of hemoglobin will cause a rise in GOT of 1 unit.

### Diseases and Injuries Causing Enzyme Elevations

Metastatic carcinoma of the liver often causes an elevation of serum enzymes. Persistent elevations of serum GOT, LDH and GPT should arouse suspicion of such a malignancy. Hill and Levi found consistent LDH elevations in patients with malignant tumors. However, the level depends on the rapidity of tumor growth (Wroblewski).

Ten percent of patients with pericarditis were found to have elevation of SGOT of 50 to 150 units on the third to the fifth day of the condition. The symptoms of pericarditis and of myocardial infarction are often identical, and differentiation of the two conditions by use of the enzyme tests might be very difficult.

Following a surgical procedure the serum enzyme level may increase considerably because of tissue injury. However, this rise is not constant. If one suspects that a patient might have sustained a myocardial infarction during or immediately after an operation, enzyme tests may be of little aid in diagnosis. Slight elevations of SGOT have been reported following cardiac catheterization and after mitral commissurotomy. Enzyme elevations were found in 72 percent of a group of patients with skeletal muscle injuries. If one suspects that a patient has sustained a myocardial infarction after an accidental injury, the enzyme tests cannot be used in the differential diagnosis.

Embolism to the lung, which causes pulmonary infarction, elevates the SGOT to 50 to 100 units in 25 to 50 percent of cases. The LDH may also be elevated. When the pain is similar to that of myocardial infarction, transient elevation of serum bilirubin or the presence of fat in the sputum may be a helpful observation pointing to a diagnosis of pulmonary infarction.

Rapid cardiac arrhythmias may cause extreme elevations of SGOT. Chinsky, Shmagranoff and Sherry reported SGOT values as high as 2,000 units following ventricular tachycardia. Because arrhythmia may give rise to chest pain similar to that of coronary occlusion, and because the patient might not be aware of a transient episode of tachycardia, an erroneous diagnosis of myocardial infarction is often made in this situation. Liver ischemia second-

ary to the tachycardia is probably the source of the increased GOT in these cases.

Because the liver is so rich in enzyme content, hepatic dysfunction is readily demonstrated by elevations of the enzymes. Hepatocellular damage in hepatitis may cause severe elevations of SGOT, GPT, and LDH. By fractionation of isozymes, Wieme found increases in the faster LDH fractions after myocardial infarction, increases in the slower fractions during periods of active hepatitis, and increases in all LDH fractions in patients with malignant tumors. A number of investigators have pointed out that measurement of GOT is a sensitive and reliable method of detecting asymptomatic carriers of hepatitis virus. In hepatitis the SGOT and GPT might be elevated to 500 to 2,000 units. Infectious mononucleosis, hepatotoxic drugs, and cirrhosis of the liver may cause elevations of the enzymes. The SGOT is elevated in 70 to 90 percent of patients with hepatic neoplasms, either primary or secondary. Cardiac decompensation and hypotension also might cause significant elevation of the enzymes due to liver ischemia. Biliary obstruction, either intrahepatic or extrahepatic, also causes elevations.

We have found that firm palpation of the liver in the dog will liberate large amounts of these enzymes into serum. If this observation is applicable to man, overly vigorous palpation of the liver during physical examination might cause a false elevation of the enzymes and lead the physician astray. Firm compression of the kidney in experimental animals also causes elevation of the enzymes but the rise is less significant.

Prolonged shock, e.g., following hemorrhage, pulmonary embolus, or coronary occlusion, may cause ischemia of the liver and give rise to an extreme elevation of all the enzymes. A study of a group of patients with hemorrhagic shock and without evidence of myocardial abnormalities showed elevation of GOT in 70 percent, elevation of LDH in 52 percent, and elevation of GPT in 37 percent. Hepatic ischemia or congestion secondary to cardiac decompensation in heart failure may result in enzyme elevations.

Infarction of the liver, kidney, spleen or gastrointestinal tract due to embolus or arterial occlusion may result in pronounced elevation of the enzymes.

Hemolysis may cause a significant rise in the enzyme levels because of the high content of the enzymes in red blood cells. Anemia has the same effect.

Many muscular diseases are likely to cause elevation of the enzymes.

## Differentiation of Coronary Occlusion and Coronary Insufficiency

Following an attack of coronary insufficiency, the enzymes are usually not elevated. Myocardial necrosis resulting from the episode usually is not sufficient to result in liberation of substantial amounts of enzymes into the plasma. Severe coronary insufficiency lasting several hours, however, may lead to significant infarction of the myocardium and to enzyme elevations.

## Conclusion

Accurate interpretation of serum enzyme tests used for differential diagnosis of myocardial infarction requires a full knowledge of the many factors which can produce elevations of the enzymes. The SGOT and LDH tests have been the most useful for diagnostic purposes. It is hoped that other enzyme and isozyme tests will also prove to be of value in the differential diagnosis of myocardial infarction.

## Acknowledgment

The technical assistance of the following is gratefully acknowledged: Willie Davis, Willis Pea, Myles Prevost, Jeanne Bloom and Katherine Wasserman.

(The figures, tables and references may be seen in the original article.)

(A Review Article, "The Clinical Significance of Serum Enzyme Estimations," by D. N. Boron MD, of the Royal Free Hospital School of Medicine, University of London, has been published in *Abstracts of World Medicine* 40:377-387, December 1966. This is a very comprehensive review. The author emphasizes, in his conclusion that it is better for a laboratory to measure a small range of enzymes accurately than a large number inaccurately and lists estimation of the serum activity of amylase, acid and alkaline phosphatases, aspartate transaminase, creatine kinase, lactate dehydrogenases, and pseudocolinesterase as those which can provide information for the clinician unobtainable by other means.—Editor.)

# SERIOUS OCULAR COMPLICATIONS ASSOCIATED WITH BLOWOUT FRACTURES OF THE ORBIT

*A. T. Milauskas MD and G. F. Fueger MD, (From the Wilmer Ophthalmological Institute and the Department of Radiology, The Johns Hopkins Hospital.)* *Amer J Ophthal 62(4):670–672, October 1966.*

The literature is replete with reports concerning the diagnosis and treatment of blowout fractures of the orbital floor. The clinical signs of vertical diplopia, enophthalmos, lid ecchymosis, emphysema and infraorbital nerve hypesthesia are well known. However, an important aspect of blowout fractures somewhat neglected in the literature is that serious ocular injuries may be present, in addition to the usual clinical signs and symptoms.

King and Samuel,[1] in 1944, in a paper entitled "Fractures of the orbit," described what is now known as a blowout fracture of the orbital floor, quoting from their description:

*I would add one other type of fracture of great importance, which is not infrequent. In this there is a downward displacement of part of the orbital floor, unassociated with any damage to the margin of the orbit or surrounding facial bones. The cause of such a fracture is difficult to visualize. The most ready explanation is trauma transmitted through the eye to the orbital floor. If, however, this were the explanation, gross damage to the eye would be expected, which is not found in most cases.*

King's hypothesis as to the pathogenesis of this type of fracture was substantiated by Smith's classical experiments on the pathogenetic mechanism of blowout fractures.[2] The present report substantiates the second part of King's hypothesis in that gross damage to the eye should also be present; indeed a significant number of patients do have ocular injuries associated with their fractures, although King did not observe this occurrence.

The relatively high incidence of ocular complications must be emphasized since this condition is not treated by ophthalmologists only; plastic surgeons, otolaryngologists and oral surgeons also repair blowout fractures of the orbital floor, and they should be aware that the possibility of a serious ocular injury exists.

In a series of 84 consecutive patients with blowout fractures or suspected of having them, seen at the Johns Hopkins Hospital, in whom the diagnosis of blowout fracture of the orbital floor was made by plain X-rays, polytomograms, orbitograms[3,4] or surgical exploration, 12 patients suffered severe ocular injuries as a result of the blunt injury that usually is the cause of blowout fracture.

## Case Reports

Case 1 (J. H. H. 34-14-38)

The patient was struck in the left eye with a fist. Clinically, there was limitation of the extraocular muscles in downward gaze and abduction; radiologically, a blowout fracture of the orbital floor was demonstrated. Vitreous hemorrhage and dislocation of the lens were also present. The lens was extracted. About one year after the injury phthisis bulbi developed and vision became no light perception.

Case 2 (J. H. H. 107-29-99)

The patient was struck in the right eye with a fist. Clinically, there was limitation of downward gaze, infraorbital nerve hypesthesia, vertical diplopia and enophthalmos. A blowout fracture of the orbital floor was demonstrated with orbitography. A traumatic cataract was seen, which rapidly matured and required extraction.

Case 3 (J. H. H. 38-93-86)

The patient had been attacked and struck in the left eye. Clinically, there was limitation of upward gaze, vertical diplopia and infraorbital nerve hypesthesia. He also had a subluxated lens and secondary glaucoma. The glaucoma has been under poor control during the two years since injury. The diplopia also persists. Initially, the diagnosis of a blowout fracture was confirmed by orbitography but surgery for its repair was refused.

Case 4 (J. H. H. 44-26-77)

The patient had been struck in the eye with a fist. Clinically, he had limitation of upward gaze, slight enophthalmos and infraorbital nerve hypesthesia.

Vision in the injured eye was reduced to the 20/70-100 range because of commotio retinae; a choroidal rupture was also present. A blowout fracture was demonstrated radiologically by orbitography. After surgical exploration, a large blowout fracture of the orbital floor was repaired. Visual acuity in the injured eye gradually became normal.

Case 5 (J. H. H. 111-50-65)

The patient had been struck in the left eye with a high-pressure water hose while working. On admission, the left eye could not be found in the socket; several examiners first thought a traumatic enucleation had occurred. The patient was taken to the operating room and the socket was explored. Most unexpectedly, a huge blowout fracture of the orbital floor was found, with total displacement of the intact globe into the left maxillary sinus. The globe was then elevated surgically and the blowout fracture repaired. Postoperatively, the visual acuity in the left eye was 20/200. A macular scar, angle recession, a post traumatic ptosis, enophthalmos and ophthalmoplegia were present.

Case 6 (J. H. H. 113-92-89)

During a fight, the patient was hit in the left eye. The patient was seen in the accident room where X-rays demonstrated a blowout fracture of the orbital floor and fracture of the orbital roof. A total anterior-chamber hyphema was present. Intraocular pressure was normal to slightly elevated. In the next two weeks, the eye developed hypotony and no light perception. A ruptured globe was suspected and confirmed by exploration. An enucleation was performed. Fracture of the orbital floor was also confirmed.

Case 7 (J. H. H. 51-05-34)

The patient had been struck in the left eye with a hammer. Examination showed marked proptosis of the globe, a total anterior-chamber hemorrhage and initial visual acuity of no light perception. X-rays demonstrated a large blowout fracture of the orbital floor and a fracture of the zygomatic arch. At surgical exploration, a large hematoma was evacuated and the blowout fracture of the floor was confirmed but not repaired. The zygomatic arch fracture was reduced. No light perception persisted and the eye became hypotonic. On exploration a posterior rupture of the globe was found and the eye was enucleated.

Case 8 (J. H. H. 41-52-49)

The patient had been struck in the left eye in a fight. Clinically, vertical diplopia with limitation on upward gaze was present; a blowout fracture was therefore suspected. A partial anterior-chamber hemorrhage persisted for 10 days. Because plain films were equivocal for a blowout fracture, an orbitogram was performed after the hyphema cleared; it was negative for a blowout fracture. The patient's diplopia subsequently cleared.

Case 9 (J. H. H. 20-70-02)

The patient had been attacked and hit in the left eye. Limitation of upward gaze was present; diplopia was not present initially because of an anterior-chamber hemorrhage of 30%-40%. Radiologically, a large blowout fracture was present. After the anterior-chamber hemorrhage had cleared, a large blowout fracture of the orbital floor was found and repaired.

Case 10 (J. H. H. 102-18-99)

The patient had injured his left eye in a sleighing accident. Radiologically, a fracture of the orbital floor and roof was present. The globe had been ruptured and subsequently required enucleation.

Case 11 (J. H. H. 52-05-09)

The patient was hit in and about the right eye; he was admitted to another service with a suspected blowout fracture. The initial plain radiographs were nonrevealing. Clinically, the patient had periorbital swelling and ecchymoses of the lids of the right eye, infraorbital nerve hypesthesia and two mm of proptosis. No diplopia was present initially and there was only minimal limitation of upward gaze. Vision in the right eye was reduced to 20/200, due to macular edema. An orbitogram showed a large blowout fracture of the orbital floor. During five days of observation, the macular edema improved and vision reached 20/50. At this time the patient began to appreciate diplopia. Surgical exploration showed a large blowout fracture of the orbital floor; it was repaired. Vision subsequently improved to 20/25.

Case 12 (J. H. H. 71-15-72)

The patient was struck in the right eye by a thrown bottle. Clinically, proptosis, diplopia and limitation of upward gaze were present. A blowout fracture of the floor was demonstrated with orbitography. With the pupil dilated the indirect ophthalmoscope revealed extensive peripheral retinal edema. No retinal tears were seen. Surgical exploration revealed a blowout fracture which was repaired.

## Summary and Conclusion

In this series of 84 cases, approximately 14% of the patients had serious ocular injuries, including rupture of the globe, vitreous hemorrhage, anterior-chamber hemorrhage, chamber-angle recession, dislocated lens, traumatic cataract, secondary glaucoma, choroidal rupture, commotio retinae, a macular scar, macular and peripheral retinal edema, in addition to blowout fracture.

Since ocular injuries may be present in patients with actual or suspected blowout fracture, it is essential that a complete ophthalmologic examination be performed, with a slitlamp examination, tonometry, ophthalmoscopy (direct and indirect) with pupil dilation, to search for ocular injuries.

## References


1. King, E. F., and Samuel, E.: Fractures of the orbit. Tr. Ophth. Soc. U.K. 64:134, 1944.
2. Smith, B., and Regan, W. F., Jr.: Blowout fracture of the orbit: Mechanism and correction of internal orbital fracture. Am. J. Ophth. 44:733, 1957.
3. Milauskas, A. T., Fueger, G. F., and Schulze, R. R.: Clinical experiences with orbitography for the diagnosis of blowout fractures of orbital floor. Tr. Am. Acad. Ophth. Otol., 70:25, 1966.
4. Fueger, G. F., Milauskas, A. T., and Britton, W. W.: Roentgenologic evaluation of orbital blowout injuries. Am. J. Roentgenol. 97:614, 1966.


# ACUTE GASTRIC ULCERS INDUCED BY RADIATION


*A. Sell and T. S. Jensen, (From the Radium Centre for Jutland [Prof. S. Kaae], Arhus, Denmark.) Acta Radiol 4(4):289–297, August, 1966.*


Gastric lesions as complications in high voltage radiotherapy have become of interest in recent years, particularly in the treatment of malignant tumours of the testis. This communication is concerned with four cases of acute gastric ulceration observed following postoperative irradiation of the lumbar lymph nodes in malignant testicular tumours.

Various forms of gastric lesions following external irradiation have been reported, especially from the Walter Reed General Hospital, Washington, by Brick (1955), Hamilton (1947), Friedmann (1959), Palmer (1948), and Warren (1942). Friedmann described four types of gastric damage in a series of about 250 malignant testicular tumours: (1) dyspepsia, (2) gastritis, (3) late chronic ulcer, and (4) acute ulcer, with or without perforation.

1. Radiation-induced dyspepsia—to be distinguished from the common roentgen 'kater' occurring during the treatment—arises 6 months to 4 years later as vague gastric symptoms without clinical or radiologic signs.

2. Radiation-induced gastritis sets in earlier than the dyspepsia, as a rule 1 to 12 months after the completion of radiotherapy, and is accompanied by radiologic evidence of spasm or stenosis of the antrum. Gastroscopy reveals smoothened mucosal folds and mucosal atrophy. The pathologic basis is fibrosis of the submucous tissue.

3. Radiation-induced ulceration has its onset from 1 month to 6 years with an average of 5 months after radiotherapy. The usual ulcer symptoms are present, but food and antacids usually afford no relief. The ulcer is radiologically indistinguishable from an ordinary ulcer; it may heal spontaneously, but submucous fibrosis generally produces antral stenosis. Friedmann recommended partial gastrectomy.

4. Unlike these late, chronic gastric sequelae, the acute, radiation-induced ulcer appears as an early complication, usually manifesting itself a month or two after the radiotherapy has been completed. This ulcer is deep and penetrates the layers of the stomach, but perforation into the peritoneal cavity is usually prevented by the omentum, the intestine, or the abdominal wall. The symptoms are severe and consist of severe epigastric pain and often gastric bleeding. Surgery is advisable before serious complications arise.

The severity and frequency of the gastric lesions depend upon the size of the dosage received by the stomach. When this exceeds 4,500 R, the incidence of gastric ulcer is 25 to 30%. The higher the dose the more serious the gastric damage leading to penetration and haemorrhage.

Friedmann has also mentioned the radiation damage sustained by other structures involved in the radiation, such as the skin, subcutaneous tissue, muscles, small and large intestine, kidneys, spinal cord, and bony tissue. Although the tolerance seems to be somewhat higher in the small and large intestine than in the stomach, ulceration as well as annular stenosis due to submucous fibrosis may arise.

Only a few other authors have described gastric complications after the treatment of the retroperitoneal lymph nodes. Sejourne (1952) reported a

case of atrophic gastritis without symptoms, which had occurred after repeated irradiation of the upper lumbar lymph nodes. Mosimann (1959) described a case of ulcer of the body of the stomach arising immediately after the completion of radiation to the lumbar lymph nodes through an abdominal field. The depth dose was not stated. Conservative treatment was tried but haemorrhage and penetration necessitated operation. Piet (1960) reported 4 cases of slight, late gastric lesions in a series of 89 irradiated testicular tumours. There is no exact statement of the depth dose, but the skin dose ranged from 2,200 to 3,000 R, delivered through 2 lumbar, convergent fields with 200 kV, lmm Cu + 2 mm Al, FSD 50 cm, field size 150 to 200 $cm^2$. Duodenal ulcer was observed in two cases within one year of the completion of radiotherapy, while deformation of the antrum accompanied by mild dyspepsia was present in two cases. These cases were diagnosed 4 years and 6 years, respectively, after the treatment. Wood et coll. (1963) reported a fatal case of necrotic ulceration of the stomach and intestine after a central dose of 3,000 R, administered in two sittings at an interval of 8 days.

The gastroscopic appearances are said to be characteristic (Palmer 1948). The irradiated area, as a rule the antrum, is transformed into an almost stiff tube, with reduced or absent peristalsis, the remainder of the gastric wall being oedematous. Where an ulcer is present, it is said to be typically deep and sharp-edged; if it heals, it does so slowly and may leave no scar.

Histologic investigations have not disclosed any changes that could distinguish radiation-induced ulcers from simple ulcers. However, it has been emphasized (Wood et coll. 1963) that irradiation produces particularly marked mucous and submucous oedema, as well as vascular changes involving proliferation of endothelial cells and thickening of the vessel walls. The submucous fibrosis and the endarteritic changes are also marked, without being specific, in late lesions following irradiation.

Animal experiments performed inter alios by Engelstad (1938) have revealed lesions following irradiation of the stomach similar to those in man.

*Present material.* All testicular tumours referred during the period March 1962 to December 1964 have been treated postoperatively with telecobalt to the para-aortic and homolateral iliac lymph nodes. A total of 38 patients were treated, distributed histologically in 18 pure seminomas and 20 non-seminomatous carcinomas. Prior to radiotherapy, all the patients had been subjected to hemicastration, but not to therapeutic or prophylactic lymphadenectomy of the lumbar nodes.

The radiation technique using telecobalt, initiated 2 to 3 weeks after the operation, comprised en bloc irradiation of the para-aortic lymph nodes through two opposed fields, one ventral and one dorsal, of 200 to 240 $cm^2$ at 80 cm FSD. The fields extended from the middle of the body of the D11 to S1, the field width being about 10 cm, determined from the lymphographic and urographic findings. Irradiation of the major parts of the kidney was avoided as far as it was possible. Only in patients with definite or probable signs of nodal invasion in relation to one kidney, was this organ included in the field. After the irradiation of the lumbar lymph nodes, the patients received irradiation through a supplementary field to the iliac nodes on the operated side.

The irradiation was administered through one field daily, with a weekly central dose of about 1,000 R and a maximum dose of about 1,100 R. As far as the seminomas are concerned, the central dose ranged from 3,500 R/4 weeks to 4,500 R/5 weeks, but with the non-seminomatous carcinomas the average dose was higher, the central dose being about 4,500 to 5,000 R in 4½ to 5 weeks, the maximum dosage about 10 to 15% higher.

This irradiation technique always involves parts of the stomach, transverse colon, and small intestine, apart from the spinal cord. The material was analysed with a view to the acute radiation effects upon the stomach, while as yet the late complications cannot be assessed owing to the short follow-up time (from 5 to 25 months) for the majority of the patients treated.

Three of the 38 treated patients exhibited manifest penetrating gastric ulcers, all of which required surgical treatment, in close relation to the completion of radiotherapy.

In the first 3 cases now reported upon, there were large ulcers that required surgery, in two because of haemorrhage and in one because of the severity of the symptoms.

Arc therapy with conventional roentgen irradiation may also lead to such a high central dose that an acute gastric ulcer results. This is exemplified by Case 4 in which the ulcer healed with scar formation during expectant treatment.

Histologic examination was carried out in all four cases, but no changes that could distinguish the radiation-induced ulcers from simple ulcers were evident.

## Discussion

The dose to the gastric mucosa ranged from 4,000 to 5,000 R/5 weeks in the first three cases of definite ulcers arising in close relation to radiotherapy. In the fourth case, treated by conventional roentgen rays, the dose in the first course of treatment was 1,600 R/54 days and in the second (last) course of treatment it was 4,800 R/24 days. These doses are of the same magnitude as those which others have reported in cases of radiation-induced ulcers. According to Friedmann the tolerance dose in the stomach is about 4,000 R during 5 to 9 weeks, a tolerance dose being taken to be the upper limit which does not entail demonstrable damage, acute or chronic.

The tolerance limit doubtless varies widely from subject to subject. Twenty-seven of the 38 patients of the present series received a central dose exceeding 4,000 R/4 to 5 weeks but only three of the group experienced acute symptoms. Five patients even received a central dose exceeding 5,000 R and had no symptoms of dyspepsia to demand further investigation. It must however be pointed out that the short follow-up periods of not more than two years, do not warrant any assessment of the total number of gastric complications.

The irradiation includes the stomach in the external irradiation of the regional lymph nodes in malignant testicular tumours. The radiosensitivity of the lymph node metastases is extremely varied, but the histologic type affords some guidance. In the case of a pure seminoma, Friedmann has given the fatal tumour dose as being between 1,500 R/14 days and 3,500 R/4 weeks, but large tumours would necessitate an increase in this dose. It is also advisable to use a minimum of 3,500 to 4,000 R/4 weeks in those instances of seminomas with considerable cellular atypia or with any suggestion of transition into non-seminomatous carcinoma. As far as the seminomas are concerned the doses would appear to be within the limit of normal gastric tolerance.

The dosage problems are on the whole unsolved in the case of non-seminomatous carcinomas. However, the fatal tumour dose appears to be considerably higher than for the seminomas, presumably in the range 4,500 to 5,000 R/35 to 40 days (Notter & Ranudd 1964). The risk of radiation damage to the stomach and intestine with this sort of dosage is quite high, and the danger should be borne in mind in weighing radiotherapy against lymphadenectomy, or possibly in combining the two in treating non-seminomatous carcinomas (Müller 1962). No random treatment series to afford a consistent answer to these problems has as yet been published.

Owing to the close relation of the retroperitoneal lymph nodes to the stomach and other radiosensitive organs, such as the spinal cord and in particular the kidneys, nothing is gained by altering the field technique, e.g. the multiple field or the arc therapy technique.

It is impossible to say whether interstitial irradiation (Seitzman et coll. 1963), using $\beta$-emitting isotopes in Lipiodol Ultrafluid (e.g. $^{124}I$, $^{131}I$) administered through the lymphatics of the spermatic cord by the usual lymphographic technique, may be useful in the prophylactic treatment of the lumbar and iliac lymph nodes. The dose will be insufficient in lymph nodes replaced by tumour tissue, as the latter does not take up the contrast medium. Interstitial radiotherapy can therefore be considered only for prophylactic purposes or in the presence of micrometastases.

The treatment of the acute radiation-induced ulcers should presumably be surgical owing to the risk of haemorrhage and perforation. This accords with the course in three of the present cases. Major palpable swelling in the epigastrium a short time after radiotherapy, possibly accompanied by dyspepsia, should not be interpreted, without further investigation, as nodal metastases, as it may represent a penetrating gastric ulcer with reactive changes in the neighboring tissues, e.g., the omentum.

## Summary

Previously reported gastric lesions produced by radiotherapy are briefly reviewed and to these the authors add four cases of gastric ulceration following the irradiation of lumbar lymph nodes associated with malignant testicular tumours. The tolerance limit of the normal gastric mucosa was found to be about 4,000 R during 5 to 9 weeks, in keeping with the experience of other authors.

(The figures and case reports omitted and references may be seen in the original article.)

# EVALUATION OF THE ROLE OF NEUROSURGICAL PROCEDURES IN THE PATHOGENESIS OF SECONDARY BRAIN-STEM HAEMORRHAGES [1]

*G. K. Klintworth (From the Department of Pathology, Duke University Medical Center, Durham, N. C., U.S.A.)* *J Neurol Neurosurg Psychiat 29(5):423–425, October, 1966.*

During the experimental investigation of the pathogenesis of mesencephalic and pontine haemorrhages associated with supratentorial expanding lesions, it became apparent that removal of the intracranial mass during a particular period was often critical to their development and accentuated their severity (Klintworth, 1965). These observations raise the question whether alleviation of intracranial pressure under comparable circumstances predisposes to secondary brain-stem haemorrhages in man. This possibility is strengthened by certain clinicopathological studies which have revealed a high incidence of lumbar puncture, pneumoencephalography, ventriculography, or neurosurgical procedure in individuals manifesting secondary brain-stem haemorrhages (van Gehuchten, 1937; Le Beau, 1943; Carrillo, 1950; Cannon, 1951; Poppen, Kendrick, and Hicks, 1962; Fields and Halpert, 1953; Cabieses, 1956).

The present study attempts to elucidate the role of neurosurgical procedures in the pathogenesis of these bulbar vascular lesions in man.

## Materials and Methods

A prospective and retrospective clinicopathological investigation was performed on over 1,200 patients with supratentorial expanding lesions. The incidence and temporal relationship of neurosurgical procedures to secondary brain-stem haemorrhages was analysed. The data reviewed were obtained from the South African Institute of Medical Research, Johannesburg, South Africa, and the Duke University Medical Center and Durham Veterans Administration Hospital, Durham, North Carolina, U.S.A.

Discrete supratentorial lesions having a maximum diameter of less than 2 cm. or a volume of less than 20 ml. were excluded from the review. Because of the difficulty in evaluating minor degrees of cerebral oedema objectively, this diagnosis was accepted only when the weight and gross appearance of the brain and the history were compatible with the diagnosis. When oedema was associated with another supratentorial condition, *e.g.*, cerebral infarction, cerebral abscess, metastatic neoplasm, the lesion was classified according to the primary affection. Recent traumatic supratentorial expanding masses were excluded from the analysis, as haemorrhages in the brain-stem of such cases may have resulted from the initial trauma.

Surgically treated patients manifesting secondary brain-stem haemorrhages were classified according to the clinical manifestations at the time of surgery as follows:

| *Type* | *Manifestations* |
|---|---|
| I | Absence of clinically manifest intracranial disease |
| II | Focal neurological symptoms and/or signs, such as epilepsy, or hemiparesis, but no overt evidence of increased intracranial pressure |
| III | Mild to moderate increased intracranial pressure with or without focal manifestations. This group includes patients with (1) lumbar spinal fluid pressures of 150-400 mm., (2) mild papilloedema |
| IV | Severe increased intracranial pressure with or without focal manifestations. This group includes patients with (1) lumbar spinal fluid pressures of more than 400 mm., (2) severe papilloedema |
| V | Moribund with an obvious intracranial expanding lesion |

## Results

Secondary brain-stem haemorrhages were associated with a wide variety of supratentorial lesions which were either large focal masses or extensive diffuse lesions (Table I). Although over half of the subjects with secondary brain-stem haemorrhages

[1] This work was supported by U.S.P.H.S. grant N.B.—06805-01.

did not experience any form of surgery, an impressive number did. A few of the latter survived postoperative periods of more than three weeks and surgery clearly had no bearing on the secondary brain-stem haemorrhages. Recent spontaneous haemorrhages were present within the tumour masses of all six patients with gliomas who manifested secondary brain-stem haemorrhages after postoperative survival periods of three months to four years. Two patients with surgically treated ruptured intracranial aneurysms died 25 and 29 days after surgery. Both had recent massive supratentorial haemorrhage and infarction secondary to second bleeds.

Most postoperative patients with secondary brain-stem haemorrhages who died within a week of surgery either underwent surgery for an intracranial lesion which was predominantly haemorrhagic or oedematous, or developed postoperative cerebral haemorrhage or oedema. In two cases the restoration of cerebral blood flow by carotid endarterectomy for carotid artery thrombosis was complicated by haemorrhage into the infarcted area.

Many patients with slowly expanding supratentorial lesions, such as cerebral gliomas or subdural haematomas and secondary brain-stem haemorrhages, did not manifest any overt increase in intracranial mass. An outstanding feature of such cases was the high incidence of craniectomy with relief of increased intracranial pressure by resection of tumour, evacuation of haematomas, lobectomy, subtemporal decompression, or ventricular drainage. These patients almost invariably underwent such procedures while comatose after manifesting a progressive increase in intracranial pressure and many were moribund before surgery. Characteristic of this group was the failure to regain consciousness following surgery and death ensuing, usually within the first 48 postoperative hours.

## Discussion

An objective evaluation of the role of neurosurgical procedures in the pathogenesis of secondary brain-stem haemorrhages is extremely difficult, as the sample of case material is derived from postmortem examination and is biased towards therapeutic failures rather than successes. Although it is clear that secondary brain-stem haemorrhages can occur in the absence of surgery, the high incidence of postoperative cases, particularly with slowly expanding supratentorial lesions, warrants consideration.

In the present investigation most secondary brain-stem haemorrhages were associated with extensive supratentorial haemorrhage or oedema either alone or in association with a neoplasm, abscess, or infarct. However, in some postoperative patients with relatively slowly expanding masses haemorrhage and oedema were inconspicuous. Subjects with such supratentorial lesions invariably underwent relief of increased intracranial pressure, sometimes only with a terminal ventricular tap, after a progressive increase in intracranial pressure and generally when comatose and manifesting hypertension and unilateral or bilateral fixed dilated pupils. Although caution generally must be exercised in extrapolating from experimental observations to clinical situations, the discovery that relief of intracranial pressure during a particular period in physiological decompensation predisposes to experimentally produced secondary brain-stem haemorrhages (Klintworth, 1965) suggests that some of the present data may represent a comparable situation in man. Such an explanation may account at least in part for the high incidence of neurosurgical procedures in patients with secondary brain-stem haemorrhages and for the well-established clinical observation that bilateral fixed dilated pupils generally indicates an ominous course in su-

TABLE I

SUMMARY OF SUPRATENTORIAL LESIONS ASSOCIATED WITH SECONDARY BRAIN-STEM HAEMORRHAGES

| *Supratentorial Lesion* | *Sample Size* | *Incidence of Secondary Brain-stem Haemorrhages* | *Cases with Secondary Brain-stem Haemorrhages* | | *Neurosurgically Treated Cases with Secondary Brain-stem Haemorrhages* | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | *Preoperative Classification* | | | | | *Postoperative Survival* | |
| | | | *Without Neurosurgery* | *With Neurosurgery* | *I* | *II* | *III* | *IV* | *V* | *Less than 1 week* | *Greater than 3 weeks* |
| Cerebral glioma | 165 | 42(25.5%) | 6(14.3%) | 36( 85.7%) | 0 | 0 | 8 | 19 | 9 | 30 | 6 |
| Metastatic tumour | 96 | 11(11.5%) | 1( 9.1%) | 10( 90.9%) | 1[2] | 0 | 0 | 3 | 6 | 10 | 0 |
| Chromophobe adenoma | 18 | 3(16.7%) | 1(33.3%) | 2( 66.7%) | 0 | 0 | 2 | 0 | 0 | 2 | 0 |
| Craniopharyngioma | 14 | 2(14.3%) | 0( 0 %) | 2(100.0%) | 0 | 0 | 1 | 1 | 0 | 2 | 0 |
| Intracerebral haemorrhage | 168 | 53(31.5%) | 45(84.9%) | 8( 15.1%) | 0 | 0 | 0 | 2 | 6 | 8 | 0 |
| Cerebral infarction (recent) | 268 | 31(11.6%) | 21(67.7%) | 10( 32.3%) | 0 | 2 | 4 | 2 | 2 | 10 | 0 |
| Cerebral abscess | 84 | 6( 7.1%) | 1(16.7%) | 5( 83.3%) | 0 | 0 | 1 | 3 | 1 | 5 | 0 |
| Subdural haemorrhage | 101 | 22(21.8%) | 14(63.6%) | 8( 36.4%) | 0 | 0 | 0 | 2 | 6 | 8 | 0 |
| Miscellaneous haemorrhage and infarct[1] | 328 | 35(10.7%) | 25(71.4%) | 10( 28.6%) | 0 | 0 | 0 | 2 | 8 | 8 | 2 |
| Total cases | 1,242 | 205(16.5%) | 114(55.8%) | 91( 44.2%) | 1[2] | 2 | 16 | 34 | 38 | 83 | 8 |

[1] This group includes supratentorial haemorrhages which were situated at multiple sites as well as those associated with recent cerebral infarction.
[2] This patient underwent a frontal leucotomy for pain due to widespread malignancy. Surgery was complicated by haemorrhage into an unsuspected metastatic tumour in the frontal lobe.

pratentorial expanding masses even with adequate surgery.

The pathogenesis of secondary brain-stem haemorrhages has yet to be fully established under controlled conditions, but available clinicopathological and experimental data strongly suggests that a combination of a large supratentorial mass, a damaged brain-stem, as by displacement, and an active circulation through the brain-stem are essential to their occurrence (Klintworth, 1965, 1966). It is clear that cerebral blood flow decreases, that the cerebral circulation time increases, and that it may be impossible to demonstrate intracranial blood vessels by angiography in subjects with severe increased intracranial pressure (Kety, Shenkin, and Schmidt, 1948; Riishede and Ethelberg, 1953; Tönnis and Schiefer, 1954, 1959; Gänshirt and Tönnis, 1956; Greitz, 1956; Horwitz and Dunsmore, 1956; Woringer, Langs, Braun, and Baumgartner, 1956; Gänshirt, 1957; Gros, Vlahovitch, and Roilgen, 1959; Löfstedt and Von Reis, 1959; Pribram, 1961; Lecuire, de Rougemont, Descotes, and Jouvet, 1962; Troupp and Heiskanen, 1963; and Heiskanen, 1963). Supratentorial decompression presumably restores cerebral blood flow and initiates haemorrhages into the brain-stem only in those cases in which the vasculature of the brain-stem has been damaged as a result of mechanical shearing and/or anoxia.

Although Cushing (1902) did not make specific reference to secondary haemorrhages in the brain-stem, it is of interest that he appreciated the danger of relieving increased intracranial pressure under circumstances similar to those outlined above. He stated: 'It must be remembered, however, that the sudden removal of pressure from the brain when the blood-pressure has been forced to considerable heights may be followed by paralysis instead of a release from the major compression symptoms. The occasion of this is readily brought out by postmortem examination, which, under such circumstances, oftentimes discloses a brain and medulla of a uniform cherry-red color, from the widespread extravasation of blood due to the multiple rupture of the minute blood vessels. The external supporting pressure of the high intracranial tension has been suddenly removed, leaving the internal or intravascular pressure too great for the strength of the vessel walls.'


## Summary

A prospective and retrospective clinicopathological investigation was performed on over 1,200 patients with supratentorial expanding lesions. Most patients manifesting secondary brain-stem haemorrhages had extensive supratentorial haemorrhage or oedema either alone or in association with a neoplasm, abscess, or infarct. Although most secondary brain-stem haemorrhages occurred in the absence of surgery, a large number of cases were postoperative. Secondary brain-stem haemorrhages were almost invariably associated with supratentorial expanding lesions in which there was either a relatively rapid increase in supratentorial mass due to haemorrhage or oedema, or increased intracranial pressure was partially or completely relieved while the patient was comatose with unilateral or bilateral fixed dilated pupils and manifesting hypertension. When viewed in the light of previous experimental data the latter cases suggest that alleviation of intracranial pressure under certain circumstances may predispose to secondary brain-stem haemorrhage in man.


# MEDICAL ABSTRACTS

## CIRCULATORY CHANGES DURING THE PAIN OF ANGINA PECTORIS, 1772-1965 —A CRITICAL REVIEW

*J. W. Roughgarden MD and E. V. Newman MD, (From the Department of Medicine, School of Medicine, Vanderbilt University, Nashville, Tennessee.) Amer J Med 41:935-946, December 1966. Circulatory Changes Associated with Spontaneous Angina Pectoris, J. W. Roughgarden, ibid, pp. 947-961.*

In the first of these two reviews, after an exhaustive search of the literature, the authors report only 20 articles listing objective data obtained during the pain of spontaneous or provoked angina pectoris. Thirty attacks in 22 patients are described in these reports. The data in these indicate that spontaneous anginal attacks uniformly are associated with relative systolic and diastolic hypertension, averaging 29 percent increase in pressure over control levels and an increase of 30 percent in pulse rate. In 10 instances, the hypertension preceded the onset of sub-

jective pain; in the others, this relationship was not observed. In only two cases were observations of intra arterial pressures and electrocardiograms obtained prior to, during, and after the relief of spontaneous anginal pain. In exertional anginal attacks, they state that by far the majority appear to be associated with relative hypertension. In the summary and conclusions, certain criticisms and suggestions for further study are listed.

The second article describes and lists data obtained during 21 attacks of unprovoked anginal pain in 10 patients observed by continuous monitoring of systolic pressures and electrocardiograms. Pulmonary artery pressures were obtained in eight attacks of unprovoked angina. The author's summary is as follows:

Spontaneous or unprovoked angina pectoris is associated with elevation of systemic and pulmonary artery pressures which preceded the onset of pain in 86 percent of cases. Increases in pulse rates are much less striking than increases in pressure prior to the onset of pain.

Relief of pain, whether spontaneous or with nitroglycerine, is associated with falling systemic and pulmonary artery pressures. In more than half the cases the pressures had returned to within 10 mm. Hg of control levels at the time pain was relieved.

The changes in pressure resemble those occurring with exertional angina, or exertion without pain in patients with coronary disease. Pulse rates are uniformly higher with exertional pain than with spontaneous pain.

The success of treatment with digitalis and/or chlorothiazides suggests that heart failure plays a part in the pathogenesis of spontaneous anginal pain.

## SURGICAL MANAGEMENT OF COMPLICATIONS TO ARTERIAL PUNCTURE

*S. E. Bergentz MD, L. O. Hansson MD, and B. Norbäck MD, (From the Department of Surgery I, Sahlgrenska sjukhuset, University of Göteborg, Göteborg, Sweden.) Ann Surg 164: 1021-1026, December 1966.*

The authors warn that arterial puncture (with or without catheterization) is not without hazards although they agree that it has become an important diagnostic step. They describe complications which developed in 38 patients following percutaneous arterial punctures during the past five years. All needed surgery in the treatment of the complications. Arterial punctures in this group were done for the following studies: coronary angiography (7), thoracic angiography (4), heart catheterization (9), femoral or pelvic angiography (4), abdominal angiography (6), carotid angiography (3), angiocardiography (4), and indwelling catheter for sampling (1).

The following points are stressed in their summary and conclusion on the basis of their experience:

1. Arterial thrombosis (13 cases) should be diagnosed and operated upon early, even if risk of gangrene seems remote.

2. Pseudoaneurysms (15 cases) may cause only mild symptoms, but recurrent partial rupture and expanding hematoma makes arterial repair important.

3. Arterio-venous fistulae (6 cases) cause subjective symptoms particularly when involving the vertebral artery (2 cases). The loud machinery murmur in the neck is very annoying for the patient and makes operative treatment necessary.

4. Hematomata under the biceps tendon after puncture of the brachial artery (3 cases) should be removed to relieve flexion contracture of the elbow. Retroperitoneal hematoma (1 case) may be massive and cause shock by perforation of the posterior wall of the external iliac artery just above the inguinal ligament.

## CAVOGRAPHY FOLLOWING PLICATION OF THE INFERIOR VENA CAVA

*J. V. Blazek MD, R. L. Clark MD, and C. S. Herron MD, (From the Johns Hopkins Hospital, Baltimore, Maryland.) Amer J Roentgen 98: 888-897, December 1966.*

The authors refer to three general groups of surgical procedures currently employed to compartmentalize the vena cava: plication with mattress sutures, with metal staples, or by external clipping. These were all designed to reduce the lumen of the inferior vena cava to multiple small channels to entrap circulatory emboli larger than 2-3 mm in size and thus prevent massive and fatal embolism, yet avoid the postoperative sequellae which have been ascribed to ligation. They report 22 patients in whom 28 vena cavographies were performed after plication during the past five years. In 12 of the patients, the plication site was patent and in 10 there was complete thrombosis following the surgical procedure. Evidence of pulmonary embolization was present in

seven including one of fatal pulmonary embolus. Ligation of the vena cava of the latter had been done following recurrent embolization after plication. In the other six of the patients with evidence of pulmonary embolization postoperatively, there was patency of the plication and in two, there was suggestion of partial or complete breakdown of the plication site.

Authors conclusion: "Plication of the vena cava does not appear to be the final solution as regards the problem of pulmonary embolization."

## DIAGNOSTIC THORACOSCOPY

*H. B. Hatch Jr. MD and P. T. DeCamp MD, (From the Section on Chest Diseases and the Department of Surgery, Ochsner Clinic, New Orleans, Louisiana.) Surg Clin N Amer 48: 1405-1410, December 1966.*

This is a report of diagnostic thoracoscopy in 50 consecutive cases of pleural effusion. The correct diagnosis was established in all. Diagnoses had not been made by other studies. There were no deaths and no serious morbidity followed the procedure.

The diagnoses established were: Neoplasm, 28 (Primary, 6; Metastatic, 22); inflammatory disease, 17; Miscellaneous, 5 (Spontaneous pneumothorax, 2; Congestive heart failure, 2; trauma, 1). The authors describe the instrument and their technique in detail and they recommend that it be used more commonly for diagnosis in cases of pleural disease with effusion.

## EXPERIENCE WITH 1,000 FIBERGASTROSCOPIC EXAMINATIONS OF THE STOMACH

*N. N. Cohen MD, R. W. Hughes Jr. MD and H. E. Manfredo MD, (From the Gastrointestinal Section of the Medical Clinic of the Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.) Amer J Dig Dis 11: 943-949, December 1966.*

This is a report of fibergastroscopic examinations of 1,000 patients examined in 15 hospitals in the Philadelphia area from August 1961 to February 1965. Actually this number was selected from the first 1,200 patients examined solely on the basis of available records (unsatisfactory examinations were not included in the calculations of reliability of diagnosis). The method of examination differed from the classic approach in that questionable areas noted in x-ray examinations were examined thoroughly before the whole stomach was examined systematically. There were 381 negative examinations. Benign ulcer was diagnosed in 262 patients, gastritis in 225, gastroenterostomies (for possible marginal ulceration) in 109 and carcinoma in seventy-nine. Hyperrugosity was noted in 52, polyps in 42, malignant ulcers in 29, marginal ulcers in 13 and there were 42 unsatisfactory examinations. Ninety-seven percent diagnostic accuracy is quoted in the report but the authors believe that 90-95 percent is probably closer to correct since the diagnosis on examination with this instrument was in doubt in 44 patients. They compare their accuracy figures with those of the roentgenologists and the gastrofibroscopists have the edge. However, throughout the report, they emphasize the importance of radiology and endoscopy as complementary examinations since their combined accuracy is greater than that of either alone. Relative merits of fibroscopic versus gastroscopic examination are discussed and the opinion is that the former is better for several reasons the most important being absence of regularly occurring blind areas, ease of passage, ready acceptance as an outpatient or office procedure, greater comfort to the patient, ease of photography. Several more are listed.

As a major complication, perforation occurred but in only three of 1,514 examinations (0.19 percent). Minor complications were bleeding of varying degrees (blood transfusion needed in one patient) and some due primarily to the scope's flexibility, the instrument curling back on itself and the tip becoming trapped in a hernial sac. It was extracted successfully with the aid of external pressure under fluoroscopic guidance.

## CHRONIC INTESTINAL ISCHEMIA: A COMPLICATION OF SURGERY OF THE ABDOMINAL AORTA

*C. Rob MD and M. Snyder MD, (From the University of Rochester School of Medicine.) Surgery 60: 1141-1145, December 1966.*

The authors describe three patients who demonstrate the interdependence of the celiac, superior mesenteric, and inferior mesenteric arterial circulations when pre-existing chronic arterial occusive disease had produced a satisfactory collateral circulation. In all, the loss of the inferior mesenteric artery as a source of collateral blood supply when it was

ligated during aortic resection resulted in chronic intestinal ischemia and symptoms of visceral angina. Continued atherosclerotic obliteration of the remaining celiac and superior mesenteric arteries so excluded the patients' ability to form an effective collateral blood flow that gradual intensification of the patients' symptoms from intestinal ischemia forced reoperation.

They stress that ligation of any one of these three arteries should be attempted only after adequacy of flow in the other two has been verified and that, when indicated at the time of aortic resection, the reconstitution of the mesenteric arterial flow by the insertion of a bypass prosthesis avoids the possible postoperative complications of chronic intestinal ischemia.

# AWARDS AND HONORS SECTION

SILVER STAR

Bjishkian, Mark E. S., HM3, USNR
Gillespie, Martin L. Jr., HM3, USN
Greer, Gerald M., HM3, USN
Leitner, Leny L., HM3, USN
Lewandowski, Michael J., HM2, USN
Mayton, James A., HM1, USN
Mullen, Thomas A., HM3, USN
Scearse, Roger D., HM3, USN
Szal, Anthony J. Jr., HM3, USN
Wiggins, Delmar J., HM3, USN
Williamson, Michael L., HN, USN

BRONZE STAR

Allen, Alvin Y. Jr., HN, USN
Barabe, William D., HM3, USN
Cessna, Ralph H., HN, USN
DeMarais, Richard D., HM3, USN
Gluechstein, William R., HN, USN
Graham, Curtis G., LT, MC, USN
Hannahan, Dennis M., HN, USN
Harrington, Robert P., HN, USN

LETTER OF COMMENDATION

Infkin, John C., LT, DC, USN (Civic Action)

VIETNAMESE MEDAL OF HONOR

Pojeky, Ruth M., CDR, NC, USN
McKay, Bernadette A., LCDR, NC, USN

NAVY COMMENDATION MEDAL

Aurelius, George M. Jr., HM3, USN (Also Bronze Star—U.S. Navy Medical News Letter 48(9), 4 November 1966.)
Drake, David L. Jr., HN, USN
Dunham, Chester, J., LCDR, MSC, USN
Gillespie, Martin L. Jr., HM3, USN
Gordon, John J., CDR, MC, USN
Roberts, James G., HM1, USN
Sampson, Donald E., LT, MC, USN
Scott, Richard B., LT, DC, USN (Civic Action)
Shaffer, Carl R., HM2, USN

# DENTAL SECTION

## IMMEDIATE DENTURE SERVICE DESIGNED TO PRESERVE ORAL STRUCTURES

*R. W. Bruce, J Pros Den 16(5): 811-821, September-October 1966.*

The article outlines a technique for producing immediate dentures based upon the following principles:

Diagnostic teamwork by the patient, prosthodontist, and the oral surgeon is very necessary in order to select only those patients adaptable to immediate dentures.

2. Immediate dentures should be planned and fabricated before *any* teeth are extracted.

3. The best time to start preserving the oral structures is at the time of extractions before oral collapse and ridge loss has occurred.

4. Most patients do not require any removal of bone or soft tissue at the time of delivery. Accepting nature's contour of the remaining ridge simplifies the denture fabrication and ultimately cuts down on surgical and postoperative problems.

5. Some undercut ridges may be improved at the

time of denture delivery by ridge recontour procedures.

6. Postoperative treatment must maintain the dentures firmly in place with good occlusion in order to provide stimulation necessary for ridge maintenance.

The procedures necessary to accomplish the desired results must begin with an accurate impression which includes the border supporting areas that show least change following extraction of the teeth.

The resulting casts are mounted on an adjustable articulator using a facebow transfer and interocclusal records. Centric relation is carefully recorded to avoid using an acquired eccentric position often found in partially collapsed mouth of immediate denture patients.

The stone teeth are cut from the casts without changing the ridge contour. The artificial teeth are arranged to give the esthetic effect that the patient's teeth had but with emphasis on *balanced occlusion.*

The dentures are processed in acrylic and remounted to the original articulator mountings for occlusal corrections.

The completed dentures are then delivered by the oral surgeon. Complete extraction of all remaining teeth under hospital conditions allows delivery of the complete dentures. The dentures can also be sectioned for delivery on an outpatient basis as a few teeth are extracted per office visit.

The surgeon is asked to try to extract the teeth without removal of bone or soft tissue. This may require sectioning of multirooted teeth. No sutures are used.

If the oral surgeon is expected to recontour an undercut ridge, a plaster cast is prepared outlining the area to be recontoured to fit the previously prepared denture. Detailed instruction for preparation of the dentures for recontour is given in the article.

The usual routine for a sectioned denture is to cut out a section of the denture leaving a hole for the remaining natural teeth to fit through. The borders of the dentures are not disturbed and the removed section is added with self-polymerizing acrylic (over a matrix) when the remaining teeth are extracted.

Soft liner (such as Hydrocast and Coe Soft) is used as soon as needed to maintain a close fit on the dentures.

After all teeth have been removed and healing is complete, the dentures are relined, rebased or remade as found necessary by the prosthodontist.

The instructions to the patient as to his need for home care and check-ups by his dentist should insure the maintenance of the maximum denture support and the least oral collapse possible for each person treated.

(Abstracted by: CAPT R. W. Bruce DC USN.)

## PERSONNEL AND PROFESSIONAL NOTES

### PERSONNEL LOSSES AT EXPIRATION OF ENLISTMENT

Historically, one of the greatest problems facing us is the loss of personnel at the expiration of their first enlistment.

These are people that are trained to provide the caliber of assistance needed to accomplish the mission of the Dental Corps. This is especially true since we are currently engaged in a broad program to provide preventive and periodontic treatment to all members of the Navy and Marine Corps. Severe losses are occurring in the DT3 and DT2 areas, and it is significant to note that these are the persons employed in the heartland of the program. They provide the major portion of the prophylaxis and stannous fluoride treatments so essential to successful progress of the program.

It is true that to some extent these losses of trained personnel are replaced by training additional numbers of dental technicians; however, formal schooling cannot and is not expected to provide the operational experience and competence which can only be gained at the operational level. Until this added experience is gained, the loss of the fully trained man will be evident in the loss of effectiveness.

Now these questions arise. What responsibilities do I, as a dental officer, have in this respect? Isn't someone at all commands assigned as the re-enlistment officer and/or petty officer? Isn't this the responsibility of the command? The answer to the last two questions is yes; however, the responsibility evolves to each member of a command to aid and assist in any way that he can. In this respect our association is unique in that by the nature of our duties we are directly assisted by a small number of personnel. This is usually one assistant who spends most of his day at our side. If, during the normal

course of the days operations, conversation can be directed toward the technician's future plans and the possibility of continuing in a naval career beyond his first enlistment, a larger percentage may decide to remain in the Navy. In the end such a move will benefit not only the individual but the Navy—the latter by reducing cost and loss of effectiveness coincident with the never ending training process.

Many factors contribute to the loss of personnel and not all of them are readily solvable at any level; however, many misconceptions may be corrected by explaining the reasons and background for hard-to-understand bureaucratic decisions and policies. In the area of explanation and counseling, we, as individuals, can accomplish the best work. Face to face talks are always better than statements written by those far removed from the sailor and his problem.

In discussing the advantages of a career in the Navy, time is well spent if you are able to express a couple of dollar and cents examples with your technician.

At the young age, when these people are first eligible for re-enlistment, twenty years into the future is a segment of time they are unable to comprehend. Therefore, the discussion must be kept in the context of now—not tomorrow.

If he is a DT3 with three plus years of service, bring to his attention that if he were to re-enlist for six years that a $1,296.00 re-enlistment bonus plus payment for unused leave would appear in his pocket immediately. If he has over four at the same pay grade, the amount increases to $1,398.00.

To approach these young people from the insurance angle is a difficult concept to get across. However, it is one that might be worth expounding on for a few moments. The CPO who does his twenty years and transfers to the Fleet Reserve at age 38, will probably live to age 72 (latest actuarial tables). He will therefore be paid $233.30 each month for the remainder of his life. Based on an expected life of 72 years, returns to him will amount to nearly $100,000. It might be added that this is a substantial return for twenty years service.

Looking at this $100,000 in another way (equity viewpoint), this figures out to $5,000 per year for each year served. The young man who does three or four years and gets out, loses equity of $15,000 and $20,000 respectively. His loss in equity is actually greater than his annual base pay.

The amounts quoted here are based on pay tables today, twenty years service, and CPO pay grade. Higher pay, additional years of service, and higher pay grades will make the examples significantly greater in monetary rewards.

These are some of the "money" examples that you might use to impress on the youngster the importance of weighing *all the factor*s in making up his mind to re-enlist or to get out.

In these days of personnel shortages, we must all devote maximum effort toward retention of trained personnel.

# PREVENTIVE MEDICINE SECTION

## NEW PUBLIC HEALTH SERVICE VACCINATION CERTIFICATE PHS 731

At its meeting in May 1965, the 18th World Health Assembly adopted the recommendation of the Committee on International Quarantine to amend the International Certificate of Vaccination or Revaccination against smallpox to indicate the origin and batch number of smallpox vaccine. The new certificate will come into force on 1 January 1967.

The Department of Defense Immunization Certificate (DD Form 737 white), when properly completed and authenticated, serves as a valid certificate of immunization *for military personnel* for international travel and quarantine purposes in accordance with Article 99, World Health Organization International Sanitary Regulations. Entries for smallpox, cholera, and yellow fever must be authenticated by the actual signature of the medical officer. PHS Form 731 (International Certificates of Vaccination, yellow) ordinarily need not be prepared for military personnel traveling abroad *unless traveling on passport*. In the event that a requirement for the use of this form for travel to or through a specific area becomes evident, this information will be disseminated appropriately.

For nonmilitary personnel traveling abroad, completion of the new PHS Form 731 will be accomplished as described below and further elaborated by BUMED Instruction 6230 series.

To be valid for international travel, smallpox vac-

cinations performed after 1 January 1967, will have to be recorded on the new certificate in PHS 731, International Certificates of Vaccination, *revised 9/66*. Any previous issues of this form may not be used and all stock on hand should be destroyed.

Smallpox vaccination certificates issued prior to 1 January 1967, shall continue to be valid for the period for which it was previously valid.

To be valid for international travel the certificate must be completed by the physician in detail, including his written signature. The origin and batch number of the smallpox vaccine shall be entered in the space provided. The traveler has the responsibility to have his certificate authenticated by physician signature and by the Department of Defense Immunization Stamp.

Supplies of PHS 731 (9-66) may be obtained from appropriate cognizance Symbol "I," Forms and Publications supply distribution points in the Navy Supply System citing stock number 0108-400-0703.—PrevMedDiv, BuMed.

## SMALLPOX VACCINE

*Morb & Mort Wkly Rpt, USDHEW, CDC, PHS 15(47):404-407, Nov 26, 1966.*

*Introduction.* Evaluation of the routine vaccination and revaccination of the population against smallpox must continue.

*The Risk of Introducing Smallpox.* Present quarantine practices offer, at best, only partial protection against the introduction of smallpox. The increased travel by U.S. nationals increases the probability of introduction from endemic areas of Asia, Africa, and South America. The potential spread of the disease is enhanced by a lack of clinical experience leading to a delay in diagnosis.

*Risk and Efficacy of Vaccination.* Data indicate that there is a risk of complications resulting from vaccination. One hundred and twenty-nine serious complications developed. From the 14 million vaccinations in 1963, practically all among primary vaccinees. The greatest rate was among those under 1 year of age. Further investigation suggested that the complication rate would be higher if adulthood was reached before the primary vaccination. The vaccine confers a high level of protection for 3 or more years with gradual waning of protection.

*Other Prophylactic Agents.* Vaccinia immune globulin and various antiviral compounds cannot replace vaccines since they are useless unless introduced shortly after exposure and confer protection for only a few weeks.

Recommendations for Smallpox Vaccination

1. *Time of Vaccination.* Schedules of vaccination appear in the triservice directive, "Immunization Requirement and Procedures", AR 40-562/BUMED-INST 6230. 1D/AFR 161.13.

2. *Site of Vaccination.* On the skin over the insertion of the deltoid or on the posterior aspect of the arm over the triceps muscle.

3. *Methods of Vaccination.*

a. Multiple Pressure: Vaccine is placed on the skin and a series of pressures is made. The site is left uncovered.

b. Jet Injection: Specifically manufactured vaccine is inoculated intradermally and the site left uncovered.

c. Other Vaccination Techniques: Any other devices which assure takes.

4. *Interpretation of Responses.* The site should be inspected 6 to 8 days after vaccination.

5. *Types of Response.*

a. Primary Vaccination. A typical Jennerian vessicle should be present.

b. Revaccination. Two responses are possible.

(1) "Major Reaction." A vesicular or pustular lesion or an area of congestion surrounding a central lesion indicates successful revaccination.

(2) "Equivocal Reaction." Any other reaction suggests revaccination should be repeated.

6. *Types of Vaccines.* Both forms afford adequate protection. The glycerinated form requires constant refrigeration and is subject to improper storage. The lyophilized vaccine is much more stable and is only vulnerable after reconstitution.

7. *Contraindications to Vaccination.*

a. If vaccination is necessary for an individual with dermatitis, vaccinia immune globulin should be administered at the same time as the vaccine.

b. Pregnant women should receive vaccinia immune globulin simultaneously with the vaccine, particularly if the woman is a primary inoculee.

c. If vaccination of patients with leukemia, lymphoma, reticuloendothelial malignancies or dysgammaglobulinemia, or those under therapy with immuno-suppressive drugs or receiving ionizing radiation is absolutely essential, vaccinia immune globulin should be administered.

8. *Vaccinia Immune Globulin (VIG).*

a. Prophylaxis. 0.3 ml/kg by intramuscular route.

b. Treatment. 0.6 ml/kg by the intramuscular route.

(1) In eczema vaccinatum, vaccinia necro-

sum, or auto-inoculation vaccinia of the eye, VIG may be effective.

(2) VIG may be helpful in treatment of severe cases of generalized vaccinia.

(3) VIG is of NO VALUE for postvaccinal encephalitis.

9. *Thiosemicarbazones.* Derivatives of thiosemicarbazones show protective effect against smallpox and therapeutic effect for individuals with severe vaccinal complications.

## COLD-WEATHER WORRY: CROUP

*By Sherwin V. Kevy MD, Consultant 6(10): 20-23, November-December 1966.*

During these breath-freezing days, a more alarming effect of cold weather on breathing can be expected—croup in children. This acute inflammation of the laryngotracheal area produces a characteristic barking cough, inspiratory stridor, sternal retraction and respiratory distress. It can proceed to complete respiratory obstruction in such short time that one must be sure beforehand of the cause, once determined.

Most cases of croup result from an infection in the larynx and trachea. Some result from other causes. All the possibilities have been listed in the accompanying table. The author started with diphtheria or "true croup." Luckily, because so many children are immunized against it, rarely is diphtheria seen in this country. It should be suspected in any child whose croup began slowly and progressed slowly for 2 or 3 days before he developed severe dyspnea, and membranous pharyngitis, laryngitis, or tracheitis. An almost identical membrane formation can occur in severe infectious mononucleosis, so this disorder must be ruled out in doubtful cases.

Unfortunately, another form of bacterial croup is not so rare as diphtheria in this country. That is epiglottitis. In fact, practically every case of bacterial croup in children between the ages of 2 and 5 years turns out to be epiglottitis. This infection is generally believed to be caused by *H. influenzae,* Group B. In a study at the Children's Hospital Medical Center in Boston, in about 10% it was caused by beta-hemolytic streptococci, and in 3%, pneumococcus. Regardless of the organism, epiglottitis begins abruptly and progresses rapidly—sometimes in as little as 9 hours—to severe respiratory obstruction because of the swollen, "cherry red" epiglottis. The child becomes increasingly restless, his throat hurts, he has difficulty in swallowing, and he may drool.

Diagnosis can easily be made by examining the child's throat; epiglottitis looks like a bright red cherry obstructing the pharynx. When examined, the child will probably be sitting and leaning forward. Better examine him in this position; if you make him lie down, his swollen epiglottis can completely cover the laryngeal opening and may start suffocating him. Be careful, too, about depressing his tongue for a better view of the epiglottis. It's not always necessary; an inflamed epiglottis usually can easily be seen. Vigorous attempts to depress the tongue can cause sudden respiratory arrest.

Children with acute epiglottitis should be hospitalized so that if a tracheotomy becomes necessary, it may be done right away. In this hospital, over the last 20 years, 34 of the 71 children brought in with acute epiglottitis needed immediate tracheotomy. In 5 of these 34, tracheotomy had been delayed too long and they were dead on arrival at the hospital. Indications for tracheotomy in this condition are the same as they are in any disease producing mechanical respiratory obstruction. Any child who is cyanotic or whose breath sounds are markedly decreased almost certainly needs immediate tracheotomy. After tracheotomy, the child should be kept on a cool atmosphere with high humidity. If crusts begin to form in the trachea, instill physiologic saline solution periodically into the tracheal cannula.

At this hospital, acute epiglottitis is treated initially with intramuscular chloramphenicol and penicillin. However, during the past 2 years, ampicillin has been used instead of chloramphenicol whenever possible. The selection of the antibiotic depends, of course, on which microorganism is causing the croup. Ampicillin, effective against both gram-positive and gram-negative microorganisms, is a good starting drug. Continue it if *H. influenzae* is causing the croup. However, if it is a streptococcus or a pneumococcus, switch to penicillin. If it is a staphylococcus, select one of the synthetic penicillinase-resistant penicillins. Steroids are not used.

### Viruses Sometimes Implicated

Recent improvements in tissue-culture techniques have helped prove definitely that certain viruses can cause croup: the influenza virus, hemadsorption virus type 2, and the adenoviruses. Viral croup commonly occurs in children between the ages of 3 months and 3 years. Unlike bacterial croup, viral croup is usually preceded by an upper respiratory

infection lasting 2 or 3 days. Viral croup does not progress with the rapidity of bacterial croup. The physical examination in viral croup will show laryngitis, tracheitis, or even laryngotracheobronchitis. Usually, the white blood cell count is normal or only slightly elevated. The child may become restless and apprehensive if the respiratory obstruction persists or becomes severe. It is stressed that respiratory obstruction so severe as to require tracheotomy is not unusual in viral croup, especially if laryngotracheobronchitis is present. Because of this it is recommended that an otolaryngologist help you determine the extent of the inflammation.

Another form of croup that should be considered with viral croup is spasmodic croup or "false croup." It used to be thought of as an allergic reaction. More recent evidence, however, indicates the cause to be inflammation, an inflammation so mild, though, as to be completely overshadowed by the respiratory distress. Increasing evidence shows that the laryngeal spasm may be triggered by and associated with a viral respiratory infection.

Spasmodic croup is common in children between the ages of 6 months and 3 years. Usually it occurs at night while the child is asleep and usually it will clear up by morning, though it may recur on one or two following nights.

For viral croup, do not prescribe antibiotics unless it worsens and secondary infection occurs. Careful observation is a most important part of the treatment.

### Managing the Child with Croup

Careful observation is important in all three forms of croup. Until you know exactly which form of croup you are dealing with, assume it is bacterial. If the disease is not progressing rapidly and respiratory obstruction is not severe, and if the parents are capable of intelligent observation and reporting, let the child stay home. However, if the disease continues to worsen and the child looks as though he may need tracheotomy, hospitalize him.

But whether at home or at the hospital, the child will probably be extremely apprehensive, so permit only truly necessary procedures.

First of all, place the child in an environment of high humidity. In the hospital, this can be done by surrounding the child with cool mist and giving him moistened oxygen when necessary. By the way, the practice of adding detergents to the oxygen is not useful at all. At home, steam may be used. Turning the hot shower on full force will easily convert the bathroom into a steam room; the mother can hold the child comfortably on her lap.

Do not sedate the child unless absolutely necessary. It seldom is. Do not give him atropine because it tends to thicken secretions, thus adding to the obstruction.

Do not give steroids. Although some laryngologists advocate them, no advantage has been found, at all, in using prednisone or dexamethasone instead of chloramphenicol. The only proved value reported is for allergic children treated with chloramphenicol and prednisone; this steroid shortens duration of symptoms more readily than chloramphenicol alone does. In nonallergic children with croup, the use of prednisone makes no difference. Dexamethasone has been tried for allergic children in a dose of 1 milligram per 10 pounds of body weight—with only moderate success.

What about syrup of ipecac? Well, this is even more controversial than the use of steroids. Syrup of ipecac's relaxing effect upon the larynx presumably results from vagal action, and, its full therapeutic effect is not achieved without vomiting. Because of this, syrup of ipecac is rarely given for croup in hospitals. If you do give it, be sure the child is closely watched for at least an hour afterwards to guard against possible aspiration of vomitus.

Let it be stressed again: Closely watch every child with croup for signs of increasing respiratory distress. Do not wait too long before advising or doing tracheotomy. Sometimes, in a tiring, restless child, early tracheotomy can prove the best conservative management.

The author mentions some of the things that are not known about croup yet, some of the intriguing questions that still need answers. Why, for instance, does croup affect boys so much more often than girls? It's usually about 2½ to 1, and in croup associated with hemadsorption virus type 2, it's about 12 to 1. Why are so many children who develop croup obese? At our hospital, about 40% are. Why does croup recur? Why will a child with acute epiglottitis either have had viral croup before, or will suffer it later on? Why, with a certain virus, will one child develop croup and another child only a mild respiratory infection? Is there a virus that causes only croup?

Croup can best be managed by insisting on the following essentials:

—high humidity
—constant watchfulness
—antibiotics (in bacterial croup)
—when necessary, tracheotomy in time.

Causes of Croup to Consider

*Bacterial*
—Diphtheria
—Epiglottitis

*Viral*
—CA virus, influenza virus, hemadsorption virus type 2
—Adenoviruses, CCA virus

*Spasmodic*
—Indirect, because of infection

*Mechanical*
—Secondary to intubation for anesthesia
—Foreign body
—Tumor

*Allergic*
—Angioneurotic edema

## ARACHNIDISM

*W. Peter Horen MS, Depart of Med, Univ of Calif Sch of Med, San Francisco, Calif, Clinical Med 73(8): 41-43, August 1966.*

Arachnidism is considered a clinical entity because of the characteristic symptoms produced by the bite of spiders of the genus *Latrodectus,* especially that of the black widow spider, *Latrodectus mactans.* Loxoscelism (caused by the bite of brown spiders of the genus *Loxosceles*), previously reported only from South America, has recently been actually or presumptively diagnosed in the United States. Bites by other species of poisonous spiders have been reported in this country, but are of little medical importance. Because arachnidism is caused by the bite of both *Latrodectus* and *Loxosceles* spiders, and the respective syndromes, treatments, and prognoses differ considerably, the proposal is made that the disease be divided into 2 clinical entities, latrodectism and loxoscelism.

A total of 615 cases of poisonous spider bites, with 38 deaths, were reported in 18 states of the United States up to the early 1940s. A recent review of 460 fatalities caused by venomous animals in the United States during the 10-year period from 1950 through 1959 indicates that 65 (14%) of these were caused by poisonous spiders.

### Spiders and Their Habits

*Lat. Rodectus mactans* is metallic black, usually with a reddish "hourglass" on the ventral aspect of the abdomen. *Loxosceles reclusa* is tan-brown, of unremarkable appearance. With legs outspread, adult black widow females measure about 11 to 12 mm. in width and adult brown spiders about 8 to 10 mm. Sub-species of *Latrodectus mactans* may be found throughout the continental United States. *Latrodectus bishopi* (the red-legged widow) is found principally in the southeastern United States including Florida, and *Latrodectus geometricus* (the gray widow) is found in Florida and southern California. Species of *Loxosceles* are also widely distributed. *Loxosceles arizonica* is indigenous to New Mexico, Arizona and western Texas, *Loxosceles divia* to southern Texas, *Loxosceles reclusa* to southeastern and central United States, and *Loxosceles unicolor* to areas of the western United States, from New Mexico and Utah to California.

The habits of *Latrodectus* and *Loxosceles* spiders are remarkably alike. They prefer dark, quiet, well-sheltered places, although *Latrodectus bishopi* often builds its irregular webs 3 to 4 feet above the ground, from palmetto to palmetto. Black widow spiders have been reported to frequent a variety of habitats ranging from permanent holes and fissures in river banks, in piles of bricks, rocks or trash, and in sheds and garages to abandoned birds' nests, shutters, rain spouts, and—especially dangerous to man—outdoor latrines. Brown spiders have been reported to prefer dry dark areas, such as fruit cellars, basements, areas beneath porches and porch furniture, as well as open fields and rocky bluffs, closets, storerooms and other places close to human habitation.

Spiders of both genera spin irregular coarse webs, and the presence of this type of web is suggestive. Other spiders, such as *Steotoda,* spin similar webs, however, and this criterion for identification should not be relied on by the laymen. Both kinds of spiders are shy and retiring, but the fact that many victims of the black widow spider were bitten in outdoor latrines suggests that this spider is somewhat aggressive. In contrast, only 1 patient with loxoscelism was bitten in a privy, and in 75% of the reported cases, the spider was found in the victim's bed or clothing.

### Some Properties of Selected Spider Venoms

The venoms of *Latrodectus* and *Loxosceles* spiders differ considerably in their effects on human victims. The black widow and other *Latrodectus* venoms are predominantly neurotoxic; their effect appears to be mainly on the spinal cord. Liver necrosis has occurred following injection of *Latrodectus mactans* venom into rats, and one investigator recently reported liver involvement in man following

the bite of the black widow. Experiments on the venom of a common European species of *Latrodectus* have shown that it is capable of causing generalized injury to the liver, kidneys, spleen, lymph nodes, thymus and adrenals by parenchymatous necrosis of blood vessels and epithelial cells, as well as affecting nervous and lymphoid tissues. A vasoconstrictor effect of this venom acting directly on the walls of blood vessels, especially those of the viscera, has been demonstrated experimentally in dogs.

The venoms of the *Loxosceles* species found in South America and in the United States have a cytotoxic effect, although systemic effects often occur concurrently. Experimental study of the effects of *Loxosceles laeta* venom on rabbits and dogs demonstrates a temporary increase in the erythrocyte sedimentation rate shortly after the venom is inoculated; marked leukopenia follows rapidly, and symptoms of shock develop. These effects are attributed to a progressive release of histamine, instead of a direct action of the venom on the victim. Postmortem examination reveals congestive hemorrhagic lesions, especially in the liver and kidneys, and a generalized vasodilatation in all animals. This is noteworthy because of the extensive damage to the kidneys found in a fatal case of loxoscelism in Argentina and the considerable renal damage occurring in a nonfatal case in the United States. Jaundice associated with loxoscelism has often been reported, particularly in the foreign literature.

The toxicity of *Latrodectus mactans* venom shows seasonal variations. In one study, venom collected in July caused greater mortality when injected into rats than that collected in October. In another study, venom collected in November was more toxic to mice than that obtained in April and May. The apparent discrepancy probably occurred because of topographic and geographic variations. Venoms in the first study came from spiders found in different localities and altitudes; those in the second came from spiders found in the same area.

## Signs and Symptoms

### Latrodectism

There is little or no local pain when a spider bites, although swelling and discoloration of the skin at the site of the bite may occur. Necrosis and other serious local effects are absent unless secondary infection occurs. Common systemic manifestations are severe pain, usually beginning 30 to 60 minutes after the bite, development of a rigid abdomen, and perspiration. Other symptoms include muscle spasms, erratic acute pain involving the abdomen, chest, back, arms and legs, and dyspnea, vomiting, nausea, insomnia, restlessness, delirium and priapism. Fever and a rapid weak pulse have also been reported, and in 1 case hemiplegia has occurred. Symptoms from the bite of *Latrodectus bishopi* have been reported for a single case occurring in Florida. An adult male, bitten on the upper left arm, developed within 3½ hours a round red eruption about the size of a dime at the site of the bite wound, a sharp burning sensation in the shoulder area, and a sensation of tightness in the chest. Speech difficulties developed 4 hours after the bite. His temperature was subnormal. Other symptoms resembled those of a typical mild case of latrodectism.

### Loxoscelism

The bite of spiders of the genus *Loxosceles* feels somewhat like a mild honeybee or ant sting, but usually severe pain develops gradually at the site of the bite over an 8 to 10-hour period. Necrosis develops typically with a central shoughing area that ulcerates deeply, sometimes exposing the muscle planes. Healing progresses slowly over an 8 to 10-week period by means of granulation and scarring. Systemic manifestations may include general malaise, chills, fever (over 104 degrees F.), fine morbilliform rash, hemolysis, hemoglobinuria, toxic nephrosis, toxic hematosis, lymphoid hyperplasia, acute laryngeal edema, asphyxiation, cyanosis, joint pains, headache, nausea, restlessness, and convulsions. One fatal case of presumed loxoscelism recently has been reported from Oklahoma.

## Treatment

A spider bite can be treated specifically through use of the correct antivenin or nonspecifically by means of symptomatic medication. Specific therapy of latrodectism consists of administration of black widow spider antivenin (Antivenin *(Latrodectus mactans),* Lyovac®, Merck Sharp & Dohme, West Point, Pennsylvania). It should always be given in cases involving children, elderly persons, and adults who show evidence of severe systemic poisoning or who have a history of hypertension. It should be emphasized that regardless of the victim's age or size, a full dose of the antivenin must be given. Spiders do not make allowances for a person's size when they bite. The antivenin is prepared from horse serum; therefore the patient's sensitivity to horse serum must be determined before it is used.

Nonspecific treatment of latrodectism depends primarily on the use of cardiotonics and muscle relaxants, including hydrotherapy, as well as occasional use of general stimulants to relieve depression and hypotension. Use of cortisone acetate, corticotropin, and other steroids is of unproven value, and 10% calcium gluconate given intravenously is considered to be of value only for temporary symptomatic relief. Recently, methocarbamol has been recommended over calcium gluconate as a muscle relaxant, to be given as follows: 10 ml. of injectable preparation given intravenously over a 5-minute period, followed by infusion of 10 ml. in 250 ml. of 5% glucose and water at a rate of 20 drops per minute. This should be followed by 4 oral doses (800 mg. each) of the drug every 6 hours for 24 hours. This medication has been reported to relieve muscle spasms and pain, headache, nausea and respiratory distress. In South America, neostigmine has been successfully used to treat latrodectism.

Specific therapy for loxoscelism is not readily available in the United States because the antivenin is manufactured in South America, and probably the demand for it is insufficient. Nonspecific therapy is primarily based on prompt use of antihistamines (i.e., hydroxyzine hydrochloride) and antibacterial medication.

Spider venom apparently is quickly absorbed and necrosis rarely occurs after the bite of a spider of the *Latrodectus species*. Consequently, local overtreatment with corrosive chemicals is to be avoided. Because spiders often reside in dirty places, and their mouthparts may be contaminated with pyogenic or anaerobic spore-forming bacteria, topical antibacterial agents may be employed to prevent infection of the bite wound.

## KNOW YOUR WORLD

### DID YOU KNOW?

That Sarawak has been declared free of smallpox as of 5 December 1966?

The only case observed in Sibu was hospitalized on 16 October 1966. Probable source of infection was Kuching where 4 cases, with 1 death, had been reported from 26 September to 2 October 1966.[1]

---

That since the latter part of November 1966 there has been an unusual increased incidence of influenza-like disease in Czechoslovakia?

This began in eastern Slovakia and is spreading to the west. There are cases in central Slovakia, on the border between Slovakia and Moravia, and in Bohemia. All age groups are affected. Two strains have been identified as virus $A_2$, probably similar to the European strains isolated since 1964 but different from $A_2$/Singapore/57.[2]

---

That approximately 1,260,000 New York residents suffer from arthritis of varying degree?

Some 330,000 are limited in activity and 70,000 are unable to work. This disease accounts for annual loss in work days per year to $52 million.[3]

---

That more than 3,000 persons will be interviewed in an 18-month study to determine factors that induce people to fall for fakes and swindlers in the health field?

The Food and Drug Administration will conduct a nationwide study in collaboration with other national and medical agencies for the protection of the public against health frauds and quackery.[4]

---

That Pennsylvania ranked first in the nation in 1965 in the total dollar value and number of contracts awarded for the construction of new sewage treatment plants?

Pennsylvania provides a yearly grant of 2% of the total costs for construction of these sewage treatment facilities toward the cost of operating the plants.[5]

---

That the Annual Report of the World Health Organization lists 150 health projects in which the WHO has assisted in 9 countries of Southeast Asia, and 177 fellowships given to health workers from regional countries and a total expenditure for 1965 of $4,436,970?

These projects include malaria eradication, tuberculosis control, vaccine production, leprosy, smallpox and cholera control, health laboratory services, health statistics, public health administration, maternal and child health, nursing, mental health, nutrition, radiation, environmental health, medical education and quality control of drugs.[6]

That about 7 million persons in the United States have various degrees of defective hearing and of this number about 125,000 are totally deaf?

This is based on the National Health Survey of July 1959 to June 1961. Males are more prone to hearing impairment.[7]

---

That brain tissue begins to die in 4 minutes and the heart stops beating in 9 minutes if the human body is without oxygen?[8]

---

That there are at present about 10,786,000 cases of leprosy in the world?

Those undergoing treatment number 1,928,000 or 18% of the estimated total. Africa has 3,868,000 cases; the Americas—358,000; Asia—6,475,000; Europe—52,000; and Oceania—33,000.[9]

---

That a program for control of Hansen's disease is underway in Texas since 1 October 1966?

The Public Health Service has granted $267,734 for a 3-year survey, which is being administered by the Texas State Health Department.[10]


REFERENCES

1. WHO Wkly Epid Recd 41(49): 634, Dec 9, 1966.
2. WHO Wkly Epid Recd 41(49): 634, Dec 9, 1966.
3. New York State Dept of Hlth Wkly Bull 19(46): 181, Nov 14, 1966.
4. USDHEW PHS Public Hlth Rpt 81(12): 1108, Dec 1966.
5. USDHEW PHS Public Hlth Rpt 81(12): 1070, Dec 1966.
6. WHO Press Release SEAR 818 of Sept 21, 1966.
7. Metropolitan Life Ins Co Stat Bull 47: 3-5, Sept 1966.
8. Science News 90(1): 14, July 2, 1966.
9. WHO Chronicle 20(11): 417-418, Nov 1966.
10. USDHEW PHS Public Hlth Rpt 81(11): 1008, Nov 1966.


# EDITOR'S SECTION

## AMERICAN BOARD OF OB-GYN

All candidates having previously passed the Part I (written) examination of this Board and have been in practice for at least eighteen months in their current locality, may apply in January or February, 1967 to take the Part II examination November 6-10, 1967. Those applications postmarked after February 28th will not be acted upon in 1967.

Application forms and Bulletins may be obtained by writing to the office of the Secretary, Clyde L. Randall, M.D., 100 Meadow Road, Buffalo, New York 14216. Prospective candidates are urged to review the current Bulletin of the Board for complete information as to the requirements and schedule for application.

Diplomates and candidates are requested to keep the Board office advised of their current address.

## AVAILABILITY OF NBC TEACHING TEAM

During the past year the U.S. Naval Medical School has developed a mobile teaching team in the medical aspects of NBC warfare. The most usual presentation consists of a five-day course at the SECRET level. Shorter and longer briefings at other security levels can be arranged. If desired, a formal examination will be given, graded on a 0-100 numerical scale.

The teaching team is completely self-contained and is now equipped with its own audio-visual equipment. User activities are required to provide:

(1) estimated number of attendees at least 2 weeks prior to team visit,

(2) a briefing space (certified by the commanding officer to meet security regulations),

(3) certification of security clearance and need-to-know of all attendees prior to first lecture,

(4) positive access control (access badges and badge racks are provided by team),

(5) classified stowage space of 3 cubic feet with access available to team security officer on day of arrival,

(6) unclassified stowage space of approximately 900 cubic feet in general proximity of briefing space,

(7) accounting data for travel and per diem expenses for 6 officers, 1 civilian instructor (GS-13) and 1 enlisted individual,

(8) garage or secure parking space for 1 Navy 2½ ton van,

(9) exercise area 1,000 yards distant from inhabited buildings and roads in use (1 hour, afternoon, 4th day) (or gas chamber),

(10) stenographic support for typing names of attendees on certificates,

(11) electric power 110V AC (total load of 340 amperes).

Funds are made available to the Commanding Officer, Naval Medical School, for equipment, supplies, printing and other support costs of the team. Funds are not furnished CO, NAVMEDSCOL for travel and per diem costs since user requests cannot be accurately forecast. Activities anticipating use of

the team should include team travel and per diem funds in budget submissions.

Requests for the team should be submitted well in advance of desired date to the Commanding Officer, Naval Medical School. Such requests should include accounting data in order that travel orders may be issued.—Public Affairs Office, NNMC, Bethesda, Md.

(Debut of NBC Teach Team at U.S. Naval School of Aviation Medicine, U.S. Naval Aviation Medical Center, Pensacola, Florida was described in U.S. Navy Medical News Letter 47(2): 27, 21 January 1966—Editor.)

## COMMITTEE IN INJURIES

Dr. Charles V. Heck, Secretary of the American Academy of Orthopedic Surgeons, announced that CAPT William S. Stryker MC USN, Chief of Orthopedic Service at the Naval Hospital, San Diego has been nominated for membership in the Committee in Injuries. It is anticipated that the appointment will be confirmed during the annual meeting of the Academy in January 1967.

The Committee's primary function is to alert the doctor, as well as non-medical personnel, on the care of the injured. To accomplish this end the Committee in Injuries sponsors regional programs for physicians on trauma and strives to improve quality of care of the injured. In addition, it assumes responsibility of continuing the program of educating the general public concerning accident prevention. Programs for non-medical personnel, presented primarily for ambulance drivers, firemen and policemen, deal in depth with rescue and survival problems as they are related to effective discharge of duties pertaining to these individuals.

Chairman of the Committee in Injuries will be Dr. Walter Hoyt of Akron, Ohio, and the Secretary will be Dr. Charles S. Neer, New York City.—U.S. Naval Hospital, San Diego, Calif. 92134.

## 7th ANNUAL AFIP LECTURES

Pathologists and other physicians interested in this specialty are invited to attend the 7th Annual Armed Forces Institute of Pathology Lectures to be held in the Post Theatre of the Walter Reed Army Medical Center on 20-24 March 1967. Sixty-five papers will be delivered by the staff of the Institute on the latest developments in pathology.

The five-day lecture series is a review and compilation of recent information in anatomic pathology (and clinical pathology methods as they apply to pathology) involving all of the various organs and body systems. The review includes common pitfalls in diagnosis, review of unusual cases, statistical data as appropriate, review of articles published or to be published by staff members, new advances in histologic techniques, application of newer histochemical, bacteriological, biochemical, immunological and toxicological methods in the daily practice of pathology.

Dr. Elson B. Helwig, Chief of the Department of Pathology, AFIP, and Course Director, says the Lectures as designed to give the busy practicing pathologist a concise combined period of instruction and review of the most recent developments at the Institute in advance of publication. This year, three interesting optional programs will be offered including, a seminar on endocrine pathology, a seminar on selected special stains as an aid to diagnosis, and a seminar on veterinary pathology.

There is no charge for the course. Applications may be obtained by writing The Director, Armed Forces Institute of Pathology, Washington, D.C. 20305.—AFIP, Washington, D.C.

## SURGERY AT SEA

## NOW

The Seventh Fleet Aircraft Carrier USS BENNINGTON (CVS-20) played a vital role in saving the life of a Nationalist Chinese sailor stricken with a perforated appendix in the South China Sea the night of December 15.

BENNINGTON, the flagship of RADM Evan P. Aurand, Commander, Antisubmarine Warfare Group ONE (ASWGRU ONE), operating in the South China Sea, received the call for help from the Republic of China destroyer NAN YANG (DD-17), which was operating with ASWGRU ONE. Admiral Aurand quickly ordered the units of his task group into action.

USS EVERSOLE (DD-789) sent a boat to NAN YANG to pick up the seriously ill sailor, Seaman Chiu Hsien-Cheng, 24, of Tainan, Formosa. Once aboard EVERSOLE, he was carried to the destroyer's landing platform where an SH-3A helicopter, piloted by CDR Davis L. Hughes, Commanding Officer of BENNINGTON-based Helicopter Antisubmarine Squadron EIGHT picked him up and airlifted him to BENNINGTON.

Navy Hospital Corpsmen were standing by on BENNINGTON's flight deck as the helo bearing Seaman Chiu touched down. Minutes later, they had rushed him to the carrier's sick bay.

Waiting in the examining room was Dr. (Lt.) Vincent A. Guardione, 28, of Springfield, Mass. When Seaman Chiu arrived, Dr. Guardione began his examination. Corpsmen took blood tests and X-rays, while other Corpsmen prepared the operating room for surgery.

The results of the tests and examinations showed Chiu had a perforated appendix. In a race against fatal peritonitis, Dr. Guardione quickly and skillfully removed the appendix. Less than two hours after Chiu had been airlifted aboard, he was lying in BENNINGTON's Sick Bay ward out of danger. Although he couldn't speak any English, Chiu was obviously very happy at the quick, expert care he received—care that had saved his life.

THEN

During World War II, I was stationed at Port Hedland, North-West Australia, as a Government Medical Officer. The Royal Flying Doctor Service supplied a De Havilland Fox Moth for me to fly. I was the only M.O. to cover about 600 miles of coastline and its hinterland from Broome to Onslow. I was attached to the three services as they had no Medical Officers in the area most of the time.

On an occasion in about 1943, I received a signal from Perth Royal Aust. Navy advising that an American tanker travelling from the Persian Gulf had aboard a case of acute appendicitis. The tanker had no charts of the Australian coast, only a school-days atlas; there was a place called COSSACK marked on the atlas. The tanker was heading for COSSACK and sulfasuxidine was being given to the patient to reduce the inflammation.

The signal asked me to give whatever help was necessary in the circumstances. It was about sunset. I flew out to sea and found the tanker about 25 miles out. As I circled it the pompoms followed me around and I hoped they were not trigger happy. The port of COSSACK was no longer in use and no people or facilities existed there. In answer to my signal of circling the ship the tanker followed me as I guided it to Point Samson where a launch met it and took off the sick sailor. From Port Samson he was driven about 15 miles to Roebourne Hospital where the theatre was ready to receive him.

I had received the Admiralty signal late in the afternoon when I was 150 miles away at Marble Bar. About 9:00 p.m. that night I removed a gangrenous appendix. The young sailor made a good recovery and was sent eventually to Perth and was thence repatriated.—From: H. G. Dicks MBBS, President, Royal Flying Doctor Service, (W.A.) Section, Perth, W.Va. To: VADM R. B. Brown MC USN. (Submitted by: LT J. T. Henderson, Code 13, BuMed.)

## ADVANCED COURSE IN NUCLEAR SCIENCE FOR MEDICAL OFFICERS (NSMO)

*Sponsored by the Defense Atomic Support Agency (DASA) at the University of Rochester, Rochester, New York.*

| *CLASS* | INCLUSIVE DATES |
|---|---|
| #20 | June 1967–July 1968 |
| *DEADLINE DATE TO APPLY* | *SECURITY CLEARANCE REQUIREMENTS* |
| 1 March 1967 | TOP SECRET |

*Mission:* It is the mission of the NSMO Course to provide the opportunity for a limited number of selected Army, Navy and Air Force Medical Officers to acquire the additional technical education needed to cope with the radiobiological problems involved in all phases of the National nuclear energy program.

*Scope:* The course provides for a review of selected portions of mathematics and physics during the refresher phase, followed by a full academic year of graduate study involving radiological physics, health physics, biological effects of radiation, evaluation of radiation hazards, environmental hygiene and toxicology, and as electives, related areas of industrial medicine and radiology. Completion of the academic phase at acceptable performance levels may lead to a Master's Degree in Radiation Biology in one year for those entering with doctoral degree, and upon completion of additional research or special studies for those not having previous professional training. The academic phase is followed by a study of practical military nuclear medicine. During the course the medical aspects of nuclear radiation over the complete range of intensity levels from low-level peacetime laboratory situations through high-level full scale warfare situations are discussed.

It is anticipated that this course will be as follows:

*PHASE I (9 weeks)—Academic Refresher*—Summer School Session, University of Rochester, Rochester, New York.

*PHASE II (10 months)—Radiation Biology*—School of Medicine and Dentistry, University of Rochester, Rochester, New York.

*PHASE III (4 weeks)—Military Nuclear Medicine*—Field Command, DASA, Sandia Base, Albuquerque, New Mexico or other appropriate installations.

*Eligibility:* The course is primarily designed for officers of the Medical Corps. However, officers of the Medical Service Corps, in very closely allied medical fields who have had some graduate work beyond the B.S. degree may also be eligible for selection.

Requests should be forwarded in accordance with BUMED INSTRUCTION 1520.10 Series and comply with the deadline date as indicated above. All requests must indicate that a security clearance of TOP SECRET has been granted to the officer requesting attendance, or that action to obtain clearance has been initiated.—Training Branch, BuMed.

## OLD HANDS GATHER

At the combined Graduation, and 25th Anniversary of Pearl Harbor Attack Memorial Ceremonies at the Hospital Corps School, San Diego a number of old hands who fought there gathered. The exceptions were LT Ben F. Dixon HC USN (Ret) of Hospital Corps Quarterly editorial renown; and seven sons, just graduated from Hospital Corps School, representing their fathers who were at Pearl Harbor during the attack.

*—Official U.S. Navy Photograph*

Persons present for Memorial Ceremonies at Hospital Corps School, U.S. Naval Hospital, San Diego, California 7 December 1966.

Front row: MMC James Stark (USS McFarland); PNC Charles R. Bisplinghoff (USS Sumner); LT (HC) Daniel F. Blum (USS St. Louis); LT (HC) Ben F. Dixon (Hospital Corps Historian); RADM H. D. Warden (USS Breese); CDR (MSC) W. W. Wilgrube (USS Solace); CWO Wayne G. Aubrey (NAS Ford Island); CWO Waid Sooter (USS Solace).

Rear Row: CAPT (MSC) Edward F. Haase (USS Houston); CDR (MSC) Joseph P. Duane (USS Pompano); CDR (MSC) R. H. Laedtke (USS Solace); HMC P. H. Palmer (USNH, Pearl Harbor); HMCM Wm. R. Frederick (USS Sumner); HMCM Trine B. Moorhead (USS Nevada); HMCS Frances W. Browning (USS California); CAPT (MC) Hugo Wagner (USS Wright); and CDR (MSC) John Garrett (USS San Francisco).

Sons of Pearl Harbor survivors—Names of fathers' duty station during attack in parentheses.

Top Row, left to right: HN James B. Johnson (Marine Barracks, P.H.); HA John A. Moore III (USS West Virginia); HN Stephen W. Stark (USS McFarland); HA James O. Bridges (Naval Base, P.H.); HA Charles J. Wager (US Army Scofield Barracks); HA Michael N. Nilssen (NAS, Ford Island); HA Frank V. Sunquist (Naval Base, P.H.).—U.S. Naval Hospital Corps School, San Diego, California 92134.

## ACKNOWLEDGMENT

In the U.S. Navy Medical News Letter 48(11): 13-15, 2 December 1966 an omission occurred on page 15 in the first column, line 3. The sentence should read: "However, if we reach the same 90% of the insects with a chemosterilant, 90 females out of 100 will be sterilized, and *not* reproduce, and in addition the 10 females that escaped treatment will be subjected to mating competition by 90 sterile males as well as 10 normal males."

DEPARTMENT OF THE NAVY

BUREAU OF MEDICINE AND SURGERY
WASHINGTON, D.C. 20390

OFFICIAL BUSINESS

PERMIT NO. 1048

POSTAGE AND FEES PAID
DEPARTMENT OF THE NAVY